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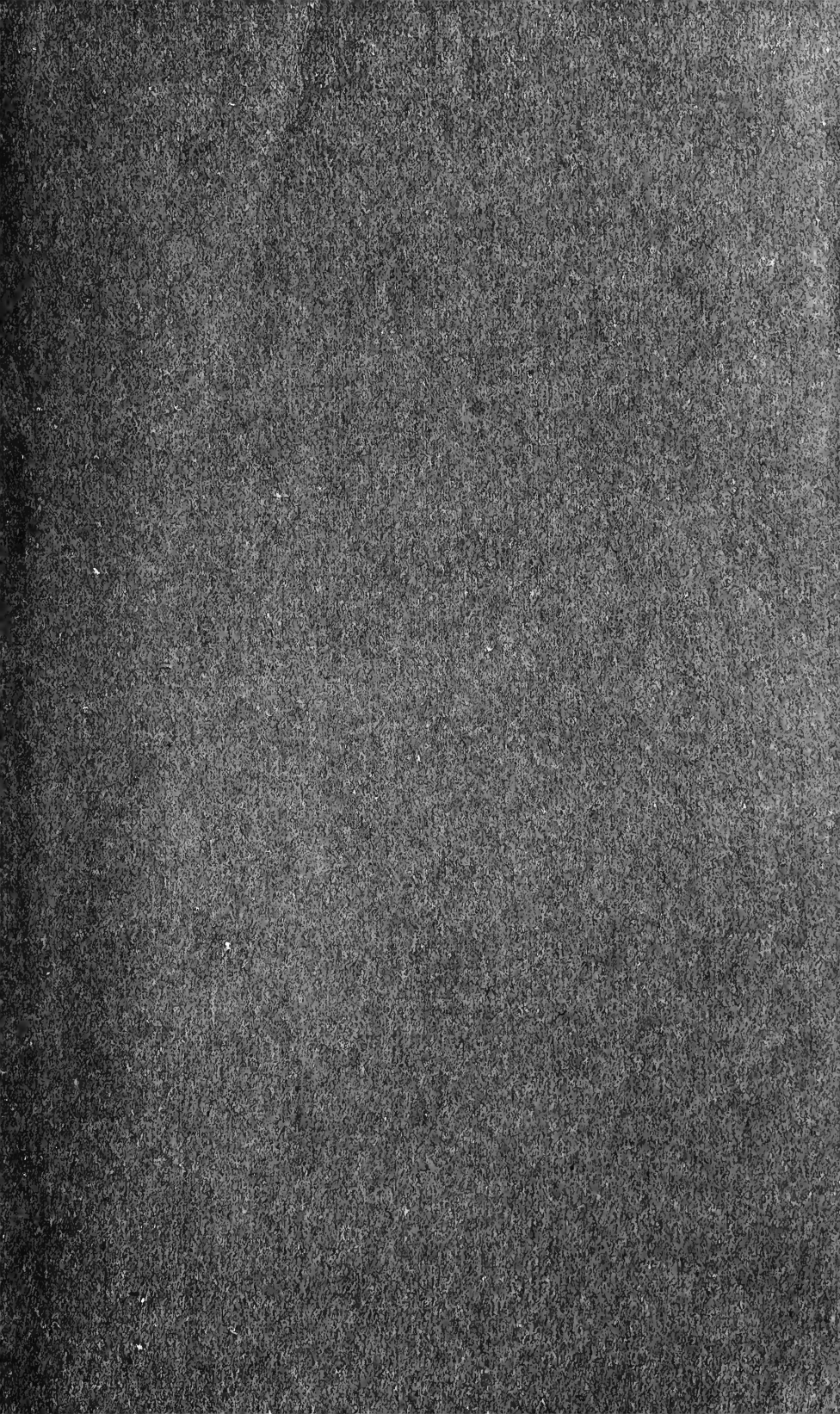
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# PULMONARY TUBERCULOSIS

BY

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ILLUSTRATED WITH 91 ENGRAVINGS AND 18 PLATES



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TO  
MY WIFE



## PREFACE.

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It is the purpose of this book to supply the general practitioner with information concerning the etiology, diagnosis, prognosis and treatment of pulmonary tuberculosis, its clinical forms and common complications. An experience of eighteen years with the tuberculosis problems in New York City has convinced the author that: (1) The physician can, and should, do more than recognize phthisis in its earliest, or pretuberculous, stage and at once send him to a sanatorium. (2) That "incipient" does not always mean *curable* tuberculosis, and conversely, that "advanced" disease does not necessarily indicate a hopeless outlook. (3) That institutional treatment is not the only effective method of handling the phthisical patient. (4) If all tuberculous persons in this country would consent to hospitalization, the available institutions would hardly accommodate ten per cent. of eligible patients. (5) Even those treated in sanatoriums must be cared for by their family physicians before admission and after discharge. (6) Careful home treatment is productive of practically the same immediate and ultimate results as institutional treatment, and is less costly to the patient and to the community.

Recent investigations of tuberculous infection have radically changed our views on the transmissibility of tuberculosis. On the one hand, it was found that patients who indiscriminately expectorate tubercle bacilli are a greater menace than has hitherto been suspected. Infants may be infected by mere contact with phthisical persons. On the other hand, there is hardly a person living in a large city who has escaped infection with tubercle bacilli. In other words, despite the vigorous and costly efforts which have been made during the past thirty years, the majority of the population in civilized countries harbor tubercle bacilli in their bodies. But, what is of more importance, not every one infected with tubercle bacilli is destined to become sick. For this reason, a sharp distinction is made in the following chapters between *infection* and *disease*, or *tuberculosis* and *phthisis*.



Recent research has also shown that infection with tubercle bacilli endows an organism with a certain degree of resistance, or even immunity, against further and renewed exogenic infection with the same virus. Experimental investigations of the past ten years have proved that it is impossible to reinfect a tuberculous animal with tubercle bacilli. Many clinical phenomena, which have hitherto baffled those who studied the disease, such as the rarity of conjugal phthisis, or of tuberculous disease in those living and working among phthisical patients, are now explained by this immunity of the tuberculous against reinfection with tubercle bacilli. Phthisis is at present considered a manifestation of immunity. Prophylaxis of *infection* has been shifted to the child, while that of phthisis involves more than prevention of infection.

In the discussion of the clinical aspects of phthisis an attempt has been made to elaborate on the constitutional symptoms, which are still the sheet anchor of the physician who is charged with deciding whether a patient is ill and in need of treatment. Bacteriology and serology are excellent helps in showing whether the patient has been infected with tubercle bacilli; skiagraphy reveals airless areas of lung tissue; but they do not give conclusive proof that the patient is sick and in need of prolonged and costly treatment. We also know that unity of causation is not always an indication of unity of resulting clinical phenomena in tuberculosis: The clinical picture of tuberculosis in infants is different from that in children; in adults some, irrespective of the treatment applied, show a marked tendency to sclerosis or fibrosis of the lesion, in others caseation and destruction of lung tissue goes on progressively; in still others there is a sluggish course, marked by periods of illness alternating with periods of comparative comfort. For these reasons several types of the disease, or syndromes, have been described, each of which has not only a different clinical course, but also a different outlook as to recovery, and the treatment differs markedly in each form of the disease.

The treatment recommended in this book is based on experience with patients in New York City. Some were living in the congested neighborhoods of the Metropolis; others in the better parts of the city; still others have been under the author's care in the hospital. A large proportion had been in sanatoriums, but even they had to be cared for in their homes before admission and after discharge. Emphasis is laid on the fact that in most cases we can give the patient the benefit of modern and approved treatment in his home as well as in institutions.

The immense utility of sanatorium treatment is emphasized and its limitations are enumerated. It is also shown that institutional treatment is not the only nor the best available method of caring for the majority of patients. Experience has taught that we can properly house and feed a patient in the city at a much less expense than in a sanatorium.

Medicinal treatment has been allotted some space for the reason that it is, in many cases, believed to possess more value than it has been accredited by therapeutic nihilists. The most recent method of treatment, artificial pneumothorax, has been given at some detail because of its efficacy in selected patients in whom everything else has failed to afford relief.

The author is under great obligations to Dr. William H. Park for reading and correcting the manuscript of the first two chapters dealing with bacteriology, a subject in which he is a master. Dr. Frederick L. Hoffman and his assistant Mr. Frederick S. Cram, of Newark, N. J., have helped with the compilation of statistical data from the recent census reports and an expression of gratitude is hardly commensurate with their labors in my behalf, and the readiness with which they have responded to my requests for data in a subject which is their own. The radiograms in this book are from plates made by Dr. Thomas Scholtz, radiographer to the Montefiore Hospital in New York City. Six have been kindly loaned to me by Dr. Charles Gottlieb, radiographer to the Beth Israel Hospital. Such excellence as may be possessed by the illustrations in the chapter on percussion is due to the skill of the well-known illustrator Lola.

M. F.

NEW YORK, 1916.





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# PULMONARY TUBERCULOSIS.

## CHAPTER I.

### THE TUBERCLE BACILLI.

THAT tuberculosis is a transmissible disease had been suspected by many ancient physicians and conclusively proved by Villemin in 1865, but it remained for Robert Koch to isolate the microorganism which is the infective agent. In 1882 he published his first communication describing the morphology, staining reactions, cultivation, and the successful animal inoculation of pure cultures of the bacilli invariably found in tissues affected with tuberculosis.

The tubercle bacillus is a parasite in the full sense of the word, living and thriving only in the bodies of animals and man, and perishing outside of the animal body. It has not been decided to which group of microorganisms it belongs; in fact, we do not as yet have a classification of bacteria which is completely satisfactory to all who are competent to judge. It may be said to belong to the group of acid-fast bacteria, of which there are many varieties to be mentioned farther on, and may be classified with the trichomycetes, while some consider it intermediary between the true bacteria and the lower fungi, the hyphomycetes.

**Morphology.**—The morphological variations of the tubercle bacilli are dependent on their type and virulence, whether human, bovine, or avian, and on the media in which they have been cultivated. In film preparations made from cultures or from sputum expectorated by tuberculous patients, the tubercle bacillus appears as a slender rod, usually straight, but very often curved, about one-fourth to one-half the diameter of a red blood corpuscle, or  $\frac{1}{500}$  mm. in length, on the average. These rods, mostly rounded on the two ends, are seen in the preparations from secretions or tissues, singly, in pairs, or in heaps, occasionally imbedded in the tissue cells. They are non-motile and have no flagella. Microscopically, an enveloping or capsular substance can often be made out around each bacillus, especially in those which have been artificially cultivated in serum for several generations. Some individual bacilli are strikingly pleomorphic, in thread, club-shaped with thickenings at either or both ends, or with filaments passing out from the main rod at right angles, and finally

in Y-shaped branchings. But these are of no practical significance because they appear to be simply degenerated types of the microorganism, although some look at them as the reverse, the result of active growth on a good culture medium and amid favorable biological surroundings. In some individual bacilli, vacuoles are seen, giving the rod the appearance of a chain of cocci. The suggestion that they represent spores appears to be erroneous, because they have no stronger resistance than the body of the bacillus, and succumb to heat and chemicals as fast as the entire rod. The fact that it is speedily killed by sunlight also indicates that the tubercle bacillus has no spores.

**Staining.**—The tubercle bacilli stain with basic dyes, but with great difficulty, and once stained they part with the color with difficulty. Their most important characteristic is their acid-fast property: While other microorganisms lose their stain when treated with acids or alcohol, the tubercle bacilli retain it. They are also alkali-fast, and when stained by an acid dye, cannot be decolorized by an alkali. But it must be mentioned that they are not the only known acid-fast bacilli. This is one of the sources of error which at times interfere with the proper appreciation of acid-fast microorganisms discovered under the microscope.

**Much's Granules.**—There have also been found tubercle bacilli which have lost their acid-fast characters while remaining virulent. Hans Much,<sup>1</sup> who has studied these microorganisms and by whose name they are generally known, describes two forms of these granules: (1) A rod-shaped granular organism; (2) isolated granules, both of which cannot be stained by the Ziehl method but only by the Gram method. They are pathogenic to animals and man and are usually found in cases of slowly progressing chronic phthisis, fibroid phthisis, cold abscess, etc. It is thus evident that before concluding that a given case lacks acid-fast bacilli, and is therefore not tuberculous, the Much granules are to be looked for by staining with the Gram-Much method. According to W. H. Park, true tubercle bacilli are probably always present together with the granules in cases in which the latter forms are found.

In this country Charles N. Meader<sup>2</sup> has recently made a careful study of these granules. In his opinion "the biological relationship of Much's forms of tubercle bacilli is a matter of considerable interest. They may be considered as a natural stage in the evolution of the bacillus, as the result of degenerative changes, or may be classed as spores (*i. e.*, as resisting forms). The accumulated evidence tends to show that they are predominantly found in tissues of a distinctly fibroid character, in old cavities, in pus of cold abscesses, in old cultures, in the notably indolent lupus lesions and in sclerosed lymph glands—facts, which taken together, mark them as forms assumed under unfavorable conditions whether they be the result of sporula-

<sup>1</sup> In Brauer, Schröder, and Blumenfeld, *Handbuch d. Tuberkulose*, vol. i, p. 193.

<sup>2</sup> *Amer. Jour. Med. Sci.*, 1915, cl, 858.

tion or of degeneration. The same conclusion is suggested by observations that under favorable cultural conditions they are rapidly replaced by Ziehl-staining forms. Against their classification as spores in the commonly accepted sense is the fact that the granular forms are rather less resistant to the action of antiformin than are the Ziehl forms; their resistance to other chemical agents has not yet been reported upon. It is of interest to note here that the granular forms appear more frequently in the bovine than in the human type of bacillus."

**Cultivation.**—The tubercle bacilli are obligatory aërobes; they require free oxygen for maintenance of life, activity, and propagation. In artificial media they grow very slowly, much slower than most bacteria which are not acid-alcohol-fast; they proliferate very slowly, and other saprophytic microorganisms which happen to live with them soon outnumber them. It is also worthy to remember that it is difficult to cultivate them directly from tuberculous lesions, secretions, and excretions of patients known to contain tubercle bacilli. But once they have been cultivated it is rather easy to transplant them to another culture of the same medium, and growth is even more luxuriant in the subsequent cultures. Theobald Smith's method of cultivation on dog serum and Dorset's egg medium are about the best and most used in this country. Pure cultures are best obtained from tubercles of animals inoculated with the bacilli. But it is often possible to obtain pure cultures from closed tuberculous cavities, from lesions of lupus, and even from sputum.

When cultivated on coagulated dog serum, or bovine serum, or in Dorset's egg medium, especially when to the latter there is added glycerin, growth appears usually at the end of ten days at 37° C., and within four weeks the characteristic growth may be expected. On the glycerin-egg medium the human form of organism produces an abundant, wrinkled layer usually having a yellowish, buff, or pinkish color. The growths are seen as more or less elevated colonies which may coalesce. On glycerin-agar the growth is more rapid than on serum, and appears as a thick, white layer, becoming yellowish. Tubercle bacilli also flourish in glycerin-potato medium.

**Powers of Resistance.**—The tubercle bacilli grow best at the temperature of the human body, 37° to 38° C., but growth is not abolished at 29° to 42° C. From a practical standpoint it is important to mention that they are not killed when exposed to moist heat of 50° C. for less than twelve hours, but heating to 55° C. for four to six hours does destroy them. They are also killed when exposed to moist heat of 60° C. for one-half hour, and in fifteen minutes at 70° C.; in five minutes at 80° to 90° C., and in one minute at 95° C. With sputum, conditions are different: the mucus protects the bacilli and it requires more time to destroy them with heat. However, five minutes' boiling is sufficient to kill the bacilli under all circumstances.

Another practical point is that in milk, tubercle bacilli resist the

action of heat with greater tenacity than in pure liquid cultures or even in sputum. From many careful experiments it appears that heating milk for thirty to forty minutes at a temperature of 65° to 70° C., or boiling for three minutes, destroys tubercle bacilli. Especially resistant are the bacilli when the milk is heated in an open vessel and a pellicle forms on the top of the fluid. This protects the bacilli against a temperature of 60° C. for an hour. William H. Park explains this by the fact that the upper parts of the fluid are not heated to the same degree as the lower and some bacilli may survive. At any rate it is important that pasteurization should be done in closed vessels. In butter the virulence of the bacilli is greatly diminished and even abolished when in contact for a long time. In fact, they die out within a few weeks as a rule. The reasons for this phenomenon are not clear. On the other hand, Schroeder and Cotton have found living tubercle bacilli retaining their virulence for one hundred and sixty days in salted butter when kept without ice in a house cellar; and Mohler, Washburn, and Doane found that they survived a year in cheese. In thoroughly boiled or roasted meat the bacilli are destroyed; but in the rare portions they may survive. Sausages, etc., made of uncooked meat, may contain living tubercle bacilli.

Dry heat is less potent in destroying tubercle bacilli; circulating steam requires one-half hour for this purpose; while bacilli in dried sputum can withstand a temperature of 100° C. for an hour. On the other hand, cold does not destroy their virulence, and freezing with subsequent thawing does not harm them very much.

It is also important to remember that the fatty substances and wax contained in the tubercle bacilli protect them to a certain extent from the effects of desiccation, and from the bactericidal action of the normal body cells, although for growth and proliferation they require moisture. When dried and pulverized by being converted into dust, as is often the case with tuberculous sputum eliminated indiscriminately by careless patients, most of the bacilli succumb, but some have been found to resist desiccation at ordinary temperature for months.

In this connection it must be borne in mind that the action of light is an important factor. It has been ascertained that light, especially sunlight, decomposes the fatty substances in the bacilli and thus destroys them altogether. When cultures are exposed to direct sunlight for a couple of hours the vitality as well as the virulence of the tubercle bacilli is destroyed; in sputum the bacilli are protected by the mucus, and it requires a longer time for their destruction. Some maintain that their virulence is only destroyed with partial loss of vitality.

Under the circumstances sputum eliminated in light places is sooner or later rendered harmless, while when expectorated in dark rooms the bacilli may retain their vitality and virulence for a year, and even drying does not harm them much.



On the whole, tubercle bacilli may retain their vitality for a considerable time if not in exceptionally unfavorable surroundings. In the latter case their growth is soon hampered, and they cannot successfully be transferred by inoculation to another culture medium; but they may retain their virulence much longer and cause disease when inoculated into an animal. After several months, however, even this wanes, and after six months this property is also lost. In laboratories it has been found by experience that it is safer to reinoculate cultures every four to six weeks. Exceptionally, cultures have been found alive and virulent after two years. This is especially the case with potato and bouillon cultures which have been kept under favorable conditions as to heat, moisture, etc., while in serum and glycerin cultures the bacilli do not survive so long.

Cornet found that serum cultures remain alive for about six months, while glycerin-agar cultures are often partially or wholly dead in six to eight weeks. There seems also to be some difference in this respect between the various types of tubercle bacilli: Maffucci states that avian bacilli may remain alive for two years, and Strauss found that cultures of human tubercle bacilli are only exceptionally capable of reproduction after five to six months; after eight to twelve months they fail regularly. Theobald Smith<sup>1</sup> recently found that a culture three months old failed, as a rule, to yield successful subcultures, and that tubercle bacilli of both human and bovine types, when kept in fully developed cultures at 40° to 50° F., may remain infectious to guinea-pigs for from seven to nineteen months, but the number of bacilli surviving in such cultures is relatively small.

The tubercle bacilli display great powers of resistance to the action of the products of other bacterial growths, in spite of the fact that they have no spores. They may survive for months in souring milk, in sewage and in-water, and in putrefying matter generally, especially sputum.

**Virulence.**—Long before the discovery of the tubercle bacillus it was known that certain diseases in animals were of the same character as human tuberculosis, and attributed to the same virus. Klenke, in 1846, emphasized the danger of milk from tuberculous cattle as an infective agent to human beings, and Villemin, in 1865, showed by animal experiment that tuberculous disease in man and animals is identical in character. With the study of the virulence of the tubercle bacillus it was found that it is pathogenic to many species of animals. In some tuberculosis is known to occur spontaneously, while others may be infected artificially. There appear to be significant differences in the results of such experimental infections, depending on the method of inoculation of the virus—injections into the subcutaneous tissues, into the peritoneum, into the anterior chamber of the eye, intravenously by feeding animals with bacilli, or compelling them to inhale the bacilli with inspired air and also according to the origin of the bacilli.

<sup>1</sup> Jour. Med. Research, 1913, xxviii, 91.

Tubercle bacilli obtained from different cases of human tuberculosis often show differences in their virulence according to the strain. But when the bacilli obtained from different animals are compared, the differences in their virulence are even more striking. For this reason there have been described different species, varieties, or stains of tubercle bacilli, although some authors maintain that the differences in cultural and virulence characteristics are acquired while the microorganisms are sojourning in the host by adaptation to the conditions favorable for their growth.

### HUMAN, BOVINE, AND AVIAN BACILLI.

The first careful study of differences in morphological, cultural, and pathogenic types of tubercle bacilli was made by Theobald Smith,<sup>1</sup> who, in 1898, showed that there are differences between the bacilli isolated from human beings when compared with those isolated from cattle. His designation of the former as "human" and the latter as "bovine" has since been generally accepted. In 1901 Robert Koch also announced that his studies led him to the conviction that human and bovine tuberculosis are not identical; that the bovine bacilli are, in fact, not pathogenic to man, and that no special measures need be taken to protect man against the consumption of milk and meat from tuberculous cattle. Considering the commercial interest which was centred around this problem, in addition to the problem of human infection, it is clear why studies along these lines have been in abundance during recent years.

Still other types of bacilli have been found. Rivolta and Maffucci have shown that there are certain morphological and biological differences between the tubercle bacilli found in birds and those in human beings. Theobald Smith continued to investigate the problem, and arrived at the conclusion that bacilli from human sources are not clearly identical in every respect with those obtained from bovine sources. Official bodies of the Imperial Department of Health in Germany, a Royal Commission in England, and Dr. William H. Park, for the New York City Department of Health, have thoroughly studied the problem, each from a different angle. The result is that we are at present in a position to state conclusively that there is more than one variety of tubercle bacillus.

The conclusions of the British Royal Commission are to the effect that "for the purposes of description it is advantageous to distinguish three types of tubercle bacilli, recognizable by their individual characters. These are the human, the bovine, and the avian. The human type, although so named, is not the only one found in cases of tuberculosis in man. It is the organism present in the majority of such cases, but in some cases of human disease the bacilli present

<sup>1</sup> Jour. of Experimental Medicine, 1898, iii, 451.

are of the bovine type, and in others the bacilli have special characters distinguishing them from each of the three principal types. In natural cases of tuberculosis in cattle the only type of bacillus present is the bovine type." William H. Park<sup>1</sup> concludes from his extensive study of the subject that "tubercle bacilli, as isolated from man, fall into two groups. One of these groups is identical in all its characters with those found in cattle. That is, all tubercle bacilli from man and cattle fall into two groups, which have been designated the human and bovine types."

**Human Bacilli.**—The human variety grows on all culture media quickly and luxuriantly; the addition of glycerin enhances their growth. On glycerin bouillon growth is seen during the first few days, and within three weeks there is seen a pellicle on the surface of the culture which spreads laterally and reaches the glass walls. The pellicle is fragile and its surface wrinkled. Morphologically, the human bacilli when grown on serum cultures appear as long, straight, or curved rods which are unevenly stained.

In general it may be stated that the virulence of human bacilli is rather low in various animals. Guinea-pigs are very susceptible and may be infected in various ways, even by rubbing the bacilli into the shaved skin of the abdomen. Rabbits are, however, less susceptible. Even when a milligram of bacilli is injected into a vein of the ear there is only produced a chronic lesion which may heal; subcutaneous injection produces an infiltration at the point inoculated which soon softens and empties itself through a fistulous opening, or may even be absorbed. The regional lymph glands swell, but do not caseate. At times, but not in every case, there may thus be produced a chronic infection of the lungs in the rabbit. Intraperitoneal inoculation produces tuberculous peritonitis, which may extend along the diaphragm; infection of the anterior chamber of the eye produces a lesion which develops more slow than when bovine bacilli are used. Cattle are infected when large doses are injected intravenously. But with subcutaneous infection there is produced only an infiltration at the point inoculated which soon suppurates and heals. The regional lymph glands swell up and at times become calcified. Feeding calves with human bacilli never produces any progressive disease. Pigs, dogs, cats, and sheep are not at all affected by human bacilli, while monkeys are very susceptible. Some species of birds are also susceptible.

**Bovine Bacilli.**—The bovine bacilli are very difficult to cultivate; it appears that the addition of glycerin to the culture medium slackens their growth. On glycerin bouillon growth is very slow. A thin pellicle is formed which spreads all over the surface within four to eight weeks, but it may remain limited to the centre of the surface. Only rarely are a few verrucose thickenings formed on the surface.

<sup>1</sup> Jour. Med. Research, 1911, xx, 313 1912, xxii, 109.

After several transplantations they may show greater tendencies to grow. Morphologically, they appear as shorter, thicker, and more evenly stained than the human variety, and usually bent, showing beading and irregularities in staining. Park, who has done excellent work along these lines, says: "Although one could in many instances make a probable diagnosis of type from an inspection of the smear, the number of intermediate gradations in morphological differences rob it of nearly all its practical value."

The bovine bacilli are more virulent for rabbits, calves and swine than the human. Guinea-pigs are just as susceptible to them as they are to the human variety, but in addition they are killed, or become acutely and progressively sick when infected with small doses of bovine bacilli. The difference in the virulence of the two types is well seen in the rabbit. The bovine type of virus causes in every instance a generalized miliary tuberculosis, progressive, and causing the death of the animal. "Human virus injected in the same amount produces either no disease at all, or lesions of varying severity in the lungs or kidneys or both, and never causes generalized miliary tuberculosis. Even with 1 mg., that is one hundred times as much, the lesions are usually confined to the same organs, and though there is a very slight tendency to generalization with this dose, there is never a generalization showing a progressive nature. Rabbits injected even with the larger dose live indefinitely, and, if death should occur the tuberculous lesions are usually not extensive enough to say that the animal died of the disease." (Park and Krumwiede.)

Cattle are also very susceptible to the bovine virus, and after intravenous injection perish from generalized tuberculosis within three or four weeks. Intraperitoneal, intra-ocular, and intramammary inoculation also cause generalized and fatal tuberculosis. Feeding cattle with even small doses of pure culture of bovine tubercle bacilli causes tuberculous disease of the intestines, followed by tuberculous lymphangitis and lymphadenitis of the mesentery; the disease spreads to other lymph glands, serous membranes, and lungs. Inhalation produces caseous pneumonia. After subcutaneous injections there is produced an infiltration at the point inoculated, swelling of the regional lymph glands and generalized tuberculosis, the animal perishing within two or three months. Pigs, sheep, goats, cats, and monkeys are very susceptible; dogs, rats, and mice are more or less refractory. Some species of birds are susceptible, but chickens show complete resistance.

**Avian Bacilli.**—On glycerin agar and on serum their growth is more luxuriant, appears more moist or slimy than observed among mammalian bacilli, and they produce an orange pigment. They grow at the temperature of 41° C. which stops the growth of mammalian tubercle bacilli. Morphologically, the differences are insignificant. The Royal Commission found that rabbits, rats, and mice are the

PLATE I

FIG. 1



Tubercle bacilli in red.  
Streptobacilli in blue.

X 1000 diameters.

FIG. 2



Tubercle bacilli in red.  
Tissue in blue.

X 1100 diameters.

FIG. 3



Leprosy bacilli in nasal secretion of person suffering from nasal lesions. (Hansen.)

X 500 diameters.

FIG. 4



Short smegma bacilli in red, rest of material in blue.

X 1100 diameters.

(From Park's Pathogenic Microorganisms.)





only mammals susceptible to inoculation with avian tubercle bacilli. Fowls are very susceptible when fed with portions of the organs containing avian bacilli, but they may consume enormous quantities of phthisical sputum without becoming tuberculous. On the other hand, the parrot is susceptible to both human and bovine bacilli as well as to avian, and spontaneous tuberculosis may be due to any of the types. Tuberculosis is very common among domesticated birds and there have been observed veritable epidemics of the disease in poultry yards.

**Tubercle Bacilli of Cold-blooded Animals.**—Certain diseases observed in worms, lizards, frogs, turtles, snakes, and fish have great resemblance to human tuberculosis and in many cases acid-fast bacilli have been isolated. These microorganisms grow luxuriantly in the room temperature, the growth being thick and moist like that of avian bacilli, and a higher temperature than 30° C. inhibits their growth. While they do not grow at the body temperature, it appears that some have been able to acclimatize them to a temperature of 36° C. Weber and Taute have cultivated this microorganism from mud and also from healthy frogs. They therefore conclude that these acid-fast bacilli have nothing in common with tubercle bacilli, but they are saprophytes which may be found in healthy animals and in the soil. Others, however, consider them as true pathogenic bacilli of cold-blooded animals, or such as have become attenuated in their virulence by a long residence in, and adaptation to growth in, a lower temperature.

Attempts have been made to use these bacilli for the purpose of immunization against infection with mammalian tubercle bacilli, but they were unsuccessful. F. F. Friedmann has even claimed that bacilli obtained from turtles are curative of existing tuberculous disease; but the results obtained have not justified in the slightest his pretensions.

**Other Acid-fast Bacilli.**—The tubercle bacilli are not the only variety of microorganisms which, once stained, refuse to be decolorized by acids and alcohol. There have been found many others presenting the same staining reactions as the tubercle bacilli and there is no doubt that they may bring about confusion in diagnosis. Of these we may mention the following:

The smegma bacillus is a slender, slightly curved rod, not unlike the tubercle bacillus but distinctly shorter, and resists the action of acids after staining. It is found in the secretions of the external genitals, mammæ, etc., especially when these secretions contain fatty matter, and there have been reported cases in which extirpation of kidneys were made mistaking these microorganisms for tubercle bacilli.

The *Bacillus lepræ* also has great similarity to the tubercle bacillus. (See Plate I.)

Moeller's grass bacilli are found in infusions of timothy-grass (*phleum pratense*), resemble morphologically the tubercle bacilli, and

are acid-fast. Inoculations produce lesions exquisitely resembling tubercles.

Moeller has also described a bacillus found in milk, even in pasteurized milk, according to Kuthy. Its similarity to the tubercle bacillus is even more pronounced than most of the other pseudotubercle bacilli. Inoculated into the peritoneal cavity of guinea-pigs, white mice, and frogs, these pseudotubercle bacilli obtained from tonsils, tongue and throat produced lesions which had great similarity, microscopically, to real tubercles, but they never spread beyond these areas. The only difference which can be discovered is that while tubercles are of a proliferative character, these pseudotubercles are of a more exudative and inflammatory character, showing a tendency to abscess formation.

Doerr and others have also isolated acid-fast rods from the excrements of cattle, swine, sheep, guinea-pigs, white mice, chickens, dogs, etc. In fact they are so frequent in the soil that any being or thing coming in contact with the soil is likely to have acid-alcohol-fast rods when carefully examined with the microscope. Doerr also found them in the dust in ordinary houses, in tap water, in centrifuge tubes, in the sediment of a laboratory flask, also in a flask of distilled water; finally in cerumal tartar on the teeth, and in the cerumen of the human ear and also in the mouth-pieces of musical instruments. He found two forms which usually occur together: One a short, thick rod, and the other a long and thin rod, very much like the tubercle bacillus. Much's stain shows usually a granular structure of the rod.

Similarly, there have been isolated microorganisms from cow's milk, butter, and from the surface of domestic animals, which morphologically, culturally, and even on inoculation resemble tubercle bacilli. The butter bacillus, first described by Petri and Rabinowitsch, may be mistaken for the tubercle bacillus even when inoculated into guinea-pigs. D. J. Davis<sup>1</sup> recently described an acid-fast streptothrix producing certain infection in the pulmonary tissues which may be mistaken for tuberculosis. Microscopically, there may be difficulty in distinguishing them, but negative results with guinea-pigs clear up the case.

It seems that the cellular structure of these pseudotubercle bacilli is closely related to that of the pathogenic tubercle bacilli, at any rate chemically, as is clearly shown by their similarity in staining reactions, and their effects locally when inoculated into animals. Some produce lesions not unlike those produced by the virulent tubercle bacilli, excepting that the general toxemia is lacking and the lesion never spreads beyond the point of inoculation. It has also been found that animals sensitized to any type of the non-virulent acid-fast bacilli, are also to some degree sensitized to the virulent form. But whether they are phylogenetically related, *i. e.*, whether they all have evolved from a common ancestry has not been established.

<sup>1</sup> Jour. Infect. Diseases, 1914, xiv, 144.



That they have not differentiated because of the variety of environment in which they have lived for many generations is proved by the fact that all efforts at making them pathogenic by passage through the bodies of various animals for several generations have failed. They always remain benign in their effect on the animal organism. The only biological characteristic they have in common with virulent tubercle bacilli are: Their acid-fast properties, and their aptitude for causing local reactions when inoculated into animals. The tubercle bacilli are alone able to produce general reactions. According to Kendal, Day, and Walker<sup>1</sup> the metabolism of the smegma and grass bacilli resembles that of the rapidly growing human bacilli. The lepra bacillus does not present this metabolic phenomenon.

It has been asked whether these acid-fast bacilli may not interfere with the diagnostic significance of the tubercle bacilli. Muir and Ritchie<sup>2</sup> thus summarize this problem: "The source of any acid-fast bacilli in question is manifestly of importance, and it may be stated that when these have been obtained from some source outside the body, or when contamination from without has been possible, their recognition as tubercle bacilli cannot be substantiated by microscopic examination alone. In the case of material coming from the interior of the body, however—sputum, etc.—the condition must be looked on as different, and although an acid-fast bacillus (not tubercle) has been found by Lydia Rabinowitsch in a case of pulmonary gangrene, we have no sufficient data for saying that acid-fast bacilli other than the tubercle bacillus flourish within the tissues of the *human* body, except in such rare instances as to be practically negligible (to this statement the case of the leprosy in bacillus is, of course, an exception). Accordingly, up until now the microscopic examination of sputum, etc., cannot be said to have had its validity shaken, and we have the results of enormous clinical experience that such examination is of practically unvarying value. Nevertheless, the facts established with regard to other acid-fast bacilli must be kept carefully in view, and great care must be exercised when only one or two bacilli are found, especially if they deviate in their morphological characters from the tubercle bacillus. In such cases inoculation may be the only reliable test."

#### OCCURRENCE OF THE VARIOUS TYPES OF TUBERCLE BACILLI.

**The Human Type.**—The human type is found in the vast majority of cases of all forms of tuberculosis in human beings; in adults phthisis is almost exclusively caused by this virus. In spontaneous tuberculosis in hogs a small percentage also shows this type of bacilli, and many species of animals, especially those coming in contact with man also are occasionally infected with human tubercle bacilli. This is

<sup>1</sup> Jour. Infect. Dis., 1914, xv, 431.

<sup>2</sup> Manual of Bacteriology, New York, 1913, p. 292.

the case with parrots and some animals in zoölogical gardens in cities, like lions, antelopes, gnu, chimpanzees, macacus rhesus, etc., that have been found infected with the human bacilli. The dog, rat, and mouse are practically immune, while the calf, rabbit, hog, and goat occupy intermediate positions.

The bovine type of tubercle bacilli is responsible for disease in domestic animals as cattle, sheep, goats, horses, etc. In most cases of tuberculosis in pigs, cats, and dogs, and in many cases in monkeys, the bovine bacilli are found.

The avian type is found in the vast majority of tuberculous infections in birds. Not only are fowls affected but also birds in zoölogical gardens are susceptible and are often sick as the result of infection with this virus. Spontaneous tuberculosis in horses, swine, monkeys, cattle, mice, and rats has been found at times to be due to this type of bacillus.

**Bovine Type of Bacillus Tuberculosis in Man.**—Of greater importance is the occurrence of bovine and avian infection in human beings. After Koch stated that the bovine bacilli were not at all identical with the human, and that they were not at all pathogenic in man, various investigations have been made with the result that Koch was, on the whole, not sustained. There is evidence to the effect that many cases of tuberculosis in human beings, especially in children, are due to the bovine virus. A large collection of reported cases was published by Park and Krumwiede, embracing 940 instances of tuberculosis carefully studied as to the type of organism present, and it appears that in adults, sixteen years of age and over, only tuberculosis of the skin, abdominal organs and general tuberculosis of alimentary origin may, at times, be caused by bovine bacilli. It is, however, a fact that but comparatively few cases have been investigated, and there is a lurking suspicion that in a larger series of cases the proportion would be much smaller. On the other hand, among 778 cases of pulmonary tuberculosis only 3, or 0.4 per cent. were found with bovine bacilli, showing conclusively that as regards phthisis, the bovine type of bacilli is not to be considered a factor in the pathogenesis of the disease.

PERCENTAGE OF INCIDENCE OF BOVINE TUBERCULOSIS IN 940 CASES OF WHICH 778 WERE PULMONARY TUBERCULOSIS (PARK AND KRUMWIEDE).

Diagnosis.	Adults 16 years and over. Per cent.	Children 5 to 16 years. Per cent.	Children under 5 years. Per cent.
Pulmonary tuberculosis . . . . .	0.4	0.0	2.8
Tuberculous adenitis, cervical . . . . .	2.7	38.0	61.0
Abdominal tuberculosis . . . . .	20.0	53.0	58.0
Generalized tuberculosis, alimentary origin . . . . .	14.0	57.0	47.0
Generalized tuberculosis . . . . .	0.0	16.0	8.6
Generalized tuberculosis including meninges, alimentary origin . . . . .	0.0	0.0	66.0
Tubercular meningitis (with or without generalized lesions other than preceding) . . . . .	0.0	0.0	4.6
Tuberculosis of bones and joints . . . . .	3.3	6.8	0.0
Tuberculosis of skin . . . . .	23.0	60.0	0.0

In children the picture is different. Under five years of age 61 per cent. of the cervical tuberculous adenitis, 58 per cent. of the abdominal tuberculosis and 66 per cent. of the generalized tuberculosis and meningitis, of alimentary origin, are caused by the bovine virus. Park's conclusions are as follows:

"Bovine tuberculosis is practically a negligible factor in adults. It very rarely causes pulmonary tuberculosis, which causes the vast majority of tuberculosis in man, and is the type of disease responsible for the spread of the virus from man to man.

"In children, however, the bovine type of tubercle bacillus causes a marked percentage of cases of cervical adenitis leading to operation, temporary disablement, discomfort, and disfigurement. It causes a large percentage of the rarer types of alimentary tuberculosis requiring operative interference, or causing the death of the child directly or as a contributing cause in other diseases.

"In young children it becomes a menace to life and causes from  $6\frac{1}{3}$  to 10 per cent. of the total fatalities from this disease."

Theobald Smith<sup>1</sup> concludes that infection with bovine bacilli in man occurs almost exclusively through the digestive tract as the portal of entry. "It has been found in the tonsils, the cervical lymph nodes, and in other organs in the generalized disease starting from these primary foci. It is probable that De Jong and Arloing isolated it from sputum. (Others have done it since.) But the fact remains that bovine infections are essentially alimentary in origin and localization, and largely restricted to childhood. A rough and liberal estimate would make from one-fourth to half the cases starting in the cervical and mesenteric lymph nodes bovine in origin."

### POISONS PRODUCED BY THE TUBERCLE BACILLI.

When tubercle bacilli enter the human body they do harm in various ways. Locally, they destroy the tissues on which they have settled, producing coagulation necrosis, etc., which will be discussed later on. By their proliferation they also produce general disturbances in the functions of the invaded body which can only be explained as caused by some poison liberated by the bacilli. The nature of these poisons is obscure at present, although strong efforts have been made to ascertain all the facts in this respect.

When dead tubercle bacilli are injected subcutaneously into the healthy animal, a distinct inflammation is produced at the site of the inoculation, frequently followed by suppuration. It is immaterial whether the bacteria have been killed by chemicals or by heat, the result is the same in either case. When dead tubercle bacilli are injected intravenously into rabbits, provided a sufficient quantity is employed for the purpose, a proliferation of tissue in the lung is

<sup>1</sup> Sixth International Congress on Tuberculosis, 1908, iv, 651.

produced similar to that of tubercles, containing, as it does, giant cells which may caseate. After intratracheal injections, tuberculous nodules with epithelioid and giant cells are produced.

On the other hand, when fluids containing the products of the metabolism of tubercle bacilli are injected in very large doses into normal and healthy animals, no toxic effects are produced.

These and other facts tend to show that the effects of the bacilli on the animal body are not due to mechanical irritation produced at the site of the inoculation, but are the result of the liberation of toxic matter which acts both locally, producing coagulation necrosis, and generally producing fever, etc. We know this, but all attempts to isolate a true toxin from tubercle bacilli have utterly failed, and with the intensive studies that have been made during the past thirty years along these lines, we have not yet been able to clearly define the tuberculous poisons. They appear to be part and parcel of the living protoplasm of the tubercle bacilli and liberated only after the latter have been destroyed. In other words, the tubercle bacilli belong to a group of microorganisms which do not secrete soluble toxins, but nevertheless produce general effects on the body which they invade; their deleterious effects are the result of the action of *endotoxins*.

**Tuberculin.**—Koch was the first to discover that when dead tubercle bacilli are injected in large quantities into *tuberculus* animals, death is caused; when small doses are injected, only a slight reaction is caused at the site of the inoculation which soon heals. On repeated inoculations he observed improvement in the condition of the sick animal. On these experimental findings he based his suggestion for the use of tuberculin as a diagnostic and therapeutic agent in tuberculosis.

Tuberculin consists mainly of the culture fluid in which the bacilli have grown, of disintegrated bacilli or extracts of their protoplasm, or both. As originally prepared by Koch, the following process is pursued:

Tubercle bacilli are cultivated on bouillon made from fresh veal to which 1 per cent. of dried peptone, 0.5 per cent. of sodium chloride, and 5 per cent. of glycerin are added. Within six to eight weeks of luxuriant growth at 38° C. the culture is poured into an evaporating dish, placed on a water bath and evaporated to one-tenth the original volume, and any remains of bacilli are removed by filtration. Containing 50 per cent. of glycerin, the resulting preparation is quite stable.

It is thus clear that tuberculin is not a true toxin, nor is it a pure endotoxin; but a 50 per cent. glycerin solution of the products of macerated tubercle bacilli in the culture fluid which are not destroyed by heat, and also any portion of bacilli which remains in the solution, or both.

Ever since the introduction of this original tuberculin, many other methods of preparation have been devised by Koch himself

and others, but all have shown that the active principle is practically the same.

**The Action of Tuberculin.**—There are differences of opinion as to whether tuberculin depends in its action on a certain chemical principle, or on several chemical substances. In fact the chemical composition of this preparation is obscure. Some have suggested that the active principle is a proteid or albumose. Klebs, Levene, and others believe that they have isolated various active principles; some have even obtained typical tuberculin reactions with these substances. But, as will be shown when discussing the tuberculin reaction, any protein inoculated into a tuberculous individual produces the same effects—tuberculosis being invariably accompanied by an altered reactivity to these substances. It can be said emphatically that at the present state of our knowledge we are in the dark as to the active principle of tuberculin.

Healthy animals bear the injection of tuberculin in large doses without any harm; the same is true of healthy human beings. Koch injected into his own body 0.25 c.c. of tuberculin and suffered from a severe reaction; after his death an autopsy showed that he had suffered from extensive pulmonary tuberculosis. On the other hand, Hamburger administered as much as 500 mgs. of tuberculin into non-tuberculous infants and children without producing the slightest local or general reaction. Clinical experience among human beings, as well as in cattle—in which it is easy and feasible to determine by autopsy whether there are tuberculous lesions—has shown that a reaction after a large dose of tuberculin in an apparently healthy person is conclusive proof of an existing tuberculous lesion somewhere in the body. We shall show later on that this is true of the vast majority of people in civilized communities, and therefore reactions to large doses of tuberculin are of very little value to the clinician who looks for active tuberculosis.

The reason why tuberculin is harmless in healthy organisms and produces such a pronounced reaction when injected into tuberculous organisms are not clear. Various theories have been advanced to explain it. The most widely accepted explanation is that of Wolff-Eisner. He assumes that tuberculous infection produces specific antibodies in the tissues which break down the tuberculin molecule, just as the digestive enzymes break down certain albumin molecules producing innocuous and highly poisonous albumoses. The antibody which acts in this manner he calls tuberculolysin. In non-tuberculous organisms there is no tuberculolysin, and when tuberculin is injected it circulates within the juices, producing no toxic effects, and is finally eliminated, like other harmless foreign proteins. In the tuberculous organism the tuberculin comes in contact with the lysin, breaks it up, and liberates a toxic substance which produces the reaction.

**Phenomena of Hypersensitiveness.**—When a rabbit is infected with tubercle bacilli, and four weeks later 0.1 to 0.3 c.c. of tuberculin is

injected subcutaneously, the animal succumbs within six to twenty-four hours. Koch found that in animals infected eight to ten weeks previously, 0.01 c.c. of tuberculin is sufficient to cause death. Injections of very small doses into tuberculous animals produce only a more or less severe reaction—fever, loss of weight, etc. This is obtained with injections of either living or dead tubercle bacilli.

When repeated small doses of tuberculin are injected, certain phenomena are observed which are not unlike those obtained after the injection of other foreign protein substances into an animal. The tuberculin reaction is evidently a manifestation of tuberculo-protein hypersensitiveness. Some authors have, indeed, been inclined to ascribe the reaction to tuberculin to the action of the non-specific substances, glycerin, proteins, extractives, etc., contained in the tuberculin and have argued that the reactions to repeated inoculations are anaphylactic phenomena. Perhaps the fact that the usual dose of tuberculin does not contain enough of foreign proteins, disproves this contention, and shows that there must be some specific substances which are active in this regard. But this has not been proved conclusively.

Theoretically, it would be expected that tuberculin, provoking the same phenomena in the animal body as the living tubercle bacilli, should also have an immunizing effect. But so far nobody has been successful in an attempt at immunization of the body with dead tubercle bacilli, or any part of the culture in which they grow. More satisfactory results have been obtained by infections with living bacilli.

Tuberculin hypersensitiveness differs from anaphylaxis by the fact that in normal animals tuberculin may be injected in large or small amounts, at long or short intervals without producing hypersensitiveness, and attempts at passive transference of tuberculin hypersensitiveness have led to doubtful results. Baldwin has been unable to produce transference, or passive anaphylaxis, from tuberculous guinea-pigs to healthy ones, and also from rabbit to rabbit, and from rabbit to guinea-pig. From human to guinea-pig the results were very doubtful, but to rabbit, partly successful. But another difference between anaphylactic shock and tuberculin hypersensitiveness may be mentioned. The former phenomenon appears immediately after an injection, while in the latter they are delayed for many hours; in the former there is a marked reduction in the temperature, etc., while in the latter the contrary is true.

**Specificity of the Tuberculin Reaction.**—We have seen that tuberculin produces obvious effects only in the infected organism. The question then arises whether the reaction it produces is strictly specific. Many workers have found that tuberculous animals react to, and may even be killed by, the injection of any foreign bacteria protein of non-tuberculous origin in the same manner as by tuberculin. In human beings there was also found hypersensitiveness to non-tuberculous

extracts from bacilli closely resembling the hypersensitiveness induced by tuberculin. Even the cutaneous tuberculin reaction can be produced by non-tuberculous toxins inoculated in the same manner as tuberculin is applied in the von Pirquet and other tests.

The changes in reactivity to tuberculin may be induced by non-tuberculous proteins and toxins. The general reaction, the fever, with concomitant subjective symptoms, such as headache, anorexia, etc., also the local reaction at the site of the inoculation and finally even the so-called "focal reaction" manifesting itself in the tuberculous lesion, have all been produced by non-tuberculous substances. On the other hand, tuberculin has produced these reactions in patients suffering from leprosy, syphilis, etc. The suggestion that this does not militate against the specificity of the tuberculin reaction because these diseases may be combined with tuberculosis, does not explain every case.

It has also been found by Mettetal<sup>1</sup> and others that individuals who react to tuberculin also react in almost the same fashion to saline solutions, which would indicate that it is not necessarily the specific bodies in the tuberculin which are responsible for the fever, malaise, etc. At any rate, tuberculin is not the only substance that produces these phenomena in tuberculous individuals.

Autopsy control has not cleared up the problem. There have been reported cases in which a positive reaction was obtained during life, but no tuberculous lesions could be discovered on careful dissection of the body after death, and the reverse. In cattle it was found that only 85 to 90 per cent. of those reacting to tuberculin show tuberculous changes on dissection after slaughter, while 10 per cent. of those which do not react show tuberculous changes in some organs. These facts have important bearings on the problems presented by tuberculin as a diagnostic agent and will be more fully discussed later on.

Another problem arises when changed reactivity to tuberculin is found. Does it invariably indicate that the body is at the time harboring living and virulent tubercle bacilli? Do individuals who have at one time passed through a tuberculous infection, but in whom the lesion has completely cicatrized also show hypersensitiveness to tuberculin? To the first question we have a positive answer—many healed cicatrized and calcified tuberculous lesions have been found to harbor virulent bacilli, as has been proved experimentally. These bacilli are in fact responsible for acute exacerbations observed in quiescent and latent tuberculosis; they may also be held responsible for the onset of the average case of phthisis in adults, as will be shown elsewhere. But what is of more importance is whether, once acquired, the tuberculin hypersensitiveness remains throughout the life of the

<sup>1</sup> Valeur de la tuberculine dans la diagnostic de la tuberculose de la première enfance, Thèse de Paris, 1900.

individual. This is a problem which has not yet been investigated to an extent as to warrant a positive answer.

Outside of these theoretical considerations, these problems have great practical bearings on the utility of tuberculin as a diagnostic agent, which is discussed in this book in its proper place.

**Mixed Infection.**—Soon after the discovery of the tubercle bacilli, some investigators, finding other pathogenic microorganisms in the secretions and excretions of phthisical subjects have maintained that the disease is due to infection with other bacteria in addition to the specific germ. In fact, many authors of ten or fifteen years ago maintained that the fever in tuberculosis is more the result of infection with pyogenic organisms than with the tubercle bacilli. The fact that contents of cavities, as well as their walls which are often covered with pyogenic membranes, often contain influenza bacilli, pneumococci, streptococci, staphylococci, etc., would tend to confirm this view. This view is even now held by many authorities. Thus Victor C. Vaughan says: "Unaided, the tubercle bacillus seldom kills, but the microbic tissue caused by its growth forms a suitable medium for the lodgement and growth of other bacteria and tuberculosis usually terminates as the result of infection. So long as the infection is unmixed, the progress of the disease is slow." But in acute miliary tuberculosis, which is invariably fatal, only the specific microorganism is found.

Recent investigations have proved conclusively that the fever in tuberculosis may be produced solely by the tubercle bacilli; indeed, an injection of tuberculin produces fever. The hectic fever of advanced phthisis, which bears great similarity to septic fever due to other causes, may also be the result of pure tuberculous activity. Even Inman, who through very laborious research found a secondary infection in all cases with fever while the patient was resting in bed, concludes that the tubercle bacillus is almost invariably the predominant infective agent.

During the course of phthisis secondary infections are often observed. A phthisical patient may be infected with pneumococci which produce pneumonia, influenza bacilli, producing grippe, etc. But this can no more be considered "mixed infection" than the association of phthisis with diphtheria, gonorrhoea, etc.

The streptococci, staphylococci, pneumococci, etc., which are often found in tuberculous cavities may, and often do, influence the symptomatology, course and termination of the disease, but in incipient cases the microorganism which is responsible for the disease is only the tubercle bacillus.



## CHAPTER II.

### TUBERCULOUS INFECTION.

**The Problems of Infection.**—With the discovery of the tubercle bacillus in 1882 it was at once concluded that practically all the problems of phthisiogenesis had been settled. The infective agent, the bacillus, enters the human body, implants itself in some tissue; by its growth and metabolic processes it produces toxic symptoms and, causing caseation and liquefaction, destroys vital organs, etc. With this knowledge, it was thought that the prevention of the disease had been reduced to simple principles: The destruction of the bacilli wherever found; and the prevention of their entry into the human body when attempts at their destruction fail for any reason.

To destroy the bacilli it was necessary to ascertain all the places where they are found in nature. This was apparently an easy matter. We know that the tubercle bacillus is a strict parasite, living and multiplying only in the human and animal body. Investigations by Sander tend to show that, within certain limits, they can proliferate on vegetable media during the hot summer months, but it is problematical whether this mode of life explains any infection in man. After the facts gathered in investigations are taken into consideration, there is no doubt that the only suitable soil for life, growth and multiplication for this bacillus is the animal body, and that the secretions and excretions of diseased persons and animals are the only means of disseminating the disease.

We have shown that bacteriologists have distinguished at least four main types of pathogenic tubercle bacilli: the human, the bovine, the avian, and the reptilian. Practical experience has shown that the last two types, those of birds and of cold-blooded animals, play no role in the epidemiology of tuberculosis in human beings, at least not a very significant role. There are consequently left the human and bovine types to be considered as etiologically important in tuberculosis in human beings.

Careful investigations by Theobald Smith, William H. Park, A. S. Griffith, Fraser, The British Royal Commission, The German Imperial Health Board, and others have shown that more than 99 per cent. of phthisis in adults and about 85 to 90 per cent. of serious tuberculous disease in children is due to the human type of bacillus; that the bovine type is found in about 10 per cent. of tuberculosis in children and in phthisis, this type is so exceptional as to make each case worthy of careful reporting. It also appears from the evidence thus far

gathered that tuberculosis in children due to bovine bacilli is mostly of the milder forms of the disease—surgical tuberculosis, of the glandular systems, especially of the thoracic and the abdominal glands, of the joints, bones, and skin. In other words, the tuberculosis caused by the ingestion of bacilli with milk from tuberculous cows is not of great significance, except perhaps in infants, when compared with the immensity of the problems presented by infections with the human type of bacilli causing phthisis in adults and fatal tuberculosis in infants.

For these reasons some authors have stated that bovine infections may be disregarded; only infection with bacilli acquired through the entry of tubercle bacilli which have been incubated, so to say, in tuberculous human beings, is to be combated, if phthisis is to be eradicated at all. The corollary to be drawn is that the sources of the tubercle bacilli are mainly human consumptives.

**Mutation of the Types of Bacilli.**—Further study has, however, complicated this problem. It has been suggested by many authors, notably Orth,<sup>1</sup> Rabinowitsch, Beitzke, Much, and others that bovine bacilli, remaining in the human body for a long time and adapting themselves to the surroundings, may acquire the characteristics of the human type, a kind of biological transformation of type, or mutation. It is clear that in our attempts at eradication of phthisis this problem is of immense importance. The 10 per cent. or more of children in civilized countries who are infected during childhood with milder forms of tuberculosis thus harbor the bovine bacilli within their bodies for many years, during which time they adapt themselves to the surroundings, and when they cause phthisis in the adult we find them with the characteristics of the human type.

In support of this assertion it was shown that very often "atypical" bacilli are found in cases of tuberculosis; they are microorganisms which cannot be classed with either the human or the bovine type. They have been called "transitional" types; types which may have been originally bovine, but after sojourning in the human body for some time are on the way to acquiring traits of human bacilli.

The British Royal Commission says in this connection that they "are inclined to regard transmutation of the bacillary type as exceedingly difficult, if not impracticable, of accomplishment by laboratory procedure; though in view of certain instances in which we obtained from one and the same human body both types of bacillus, we are not prepared to deny that transmutation of one type into another may occur in nature." Theobald Smith<sup>2</sup> also says that "the time has not yet come for us to state positively that one type cannot be transformed into another." Arloing, Marcus Robinowitsch, Sorgo, Musemeier, Dammann, and others have in fact been able to produce changes in the morphological and cultural characters, and in the virulence of

<sup>1</sup> Drei Vorträge über Tuberkulose, Berlin, 1913.

<sup>2</sup> Sixth Intern. Congr. Tuberc., 1908, iv, 651.

bacilli by passage through various animals, or cultivating them in different media. But Park and Krumwiede<sup>1</sup> say: "We have carefully examined the reports of numerous workers on this point, and cannot admit that the evidence for the transformation of type is complete." Theobald Smith, after studying the evidence also arrives at the conclusion that "in general the results of these passages have been negative so far as any recognizable modification of type is concerned." Park's suggestion that the change in type observed after passing through a series of animals is due to additional bovine infection, has a great deal in its favor. Cases in which both types were found in human beings have been reported.

We are therefore justified in concluding with Park and Krumwiede that "the two types are probably different, due to residence in different hosts over long periods of time, and as such are stable. The evidence of rapid change is incomplete and inconclusive." In the human disease the stability of type is apparently beyond question. Some cases have been followed for long years and the type of the bacillus has been found to be unaltered. Weber and Steffenhagan have followed for ten and a half years a case of surgical tuberculosis and always found bovine bacilli, without changing their typical characteristics.

The weight of evidence is thus in favor of human phthisis being due almost exclusively to human bacilli, and that infection during childhood with bovine bacilli cannot be held responsible for phthisis in the adult, because it has not been proved that mutation of one type into another takes place.

The main source of the bacilli causing phthisis in the adult, and serious or fatal tuberculosis in infants or children, appears to be the tuberculous man who expectorates myriads of bacilli fit for entering healthy persons and causing disease.

**The Channels of Entry of the Tubercle Bacilli.**—It is obvious that in order to prevent phthisis, the ways in which the bacilli enter the human body must be known definitely. To the average person, lay or medical, who has informed himself from current popular literature, this question has been answered satisfactorily: if the bacilli are derived from human sources, they have usually been inhaled; if from bovine sources they have been ingested.

But it may be stated without fear of meeting contradiction from competent sources that this problem has not yet been solved to the satisfaction of all who are entitled to an opinion. Römer,<sup>2</sup> one of the most active experimental workers in the field of tuberculosis, and one of those best qualified to speak, says that none of the given channels of entry of the tubercle bacilli is alone sufficient to adequately solve all and the complete problems of tuberculous infection.

There are three evident portals of entry which are always mentioned

<sup>1</sup> Trans. Sixth Ann. Meet. Nat. Assn. Study and Preven. Tuberc., 1910, p. 332; Jour. Med. Research, 1911, xx, 313; 1912, xxii, 109.

<sup>2</sup> In Brauer, Schröder, and Blumenfeld's *Handbuch der Tuberkulose*, 1914, i, 247.

as possible: Inhalation through the respiratory passages; ingestion through the digestive tract; inoculation into the skin or mucous membranes. While each of the three modes of infection has been shown to be possible, and proved experimentally and clinically, the inhalation channel has been considered the most important in the case of human phthisis. Ingestion may, however, be found of greater importance than it is now considered.

**Contact Infection.**—The inoculation of the tuberculous virus into the skin and mucous membranes may cause disease. This has been proved beyond any doubt both experimentally and clinically. Inoculated tuberculosis is most virulent during infancy; the younger the child, the more serious the outcome. The “pathologist’s wart” and the “butcher’s wart” in the adult are not very malignant diseases, while infection of the wound during ritual circumcision is almost invariably fatal, as has been observed by many authors, and more recently by Holt.<sup>1</sup> The reasons for these differences in virulence will be discussed later on.

Sputum from tuberculous patients is infective in another way. It enters the circulation through an abrasion. In overcrowded and filthy homes, where children creep around on the floors on which consumptives have expectorated, this mode of infection is undoubtedly quite frequent. Baldwin<sup>2</sup> and others found virulent tubercle bacilli on the fingers, and under the nails of consumptives, as well as of those who live with them. It has also been established that under exceptional circumstances infection is possible through the unbroken skin of animals.

Skin infections produce local lesions at the point of entry of the bacilli, and in infancy a fatal bacteremia may be the result. But when cutaneous skin affections such as lupus vulgaris, tuberculosis verrucosa cutis, or the so-called tuberculides are considered, it must not be concluded that they have invariably been acquired by local infection. As will be shown later on, while discussing the aërogenic and hematogenic origin of tuberculosis, the bacilli may have been brought to the skin by the blood stream. This has in fact been found in most cases. It is noteworthy in this connection that phthisis occurs only exceedingly rarely in patients with lupus and other tuberculous skin affections. Some have spoken of an immunity against tuberculosis possessed by these patients. The reverse also appears to be true—tuberculous skin disease is rare in phthisical patients, and when we bear in mind the opportunities for infection we are justified in speaking of immunity.

On the whole it appears that Römer is on a sound foundation when he says that the problem of infection through the skin has not yet been studied sufficiently, and with our present knowledge we are

<sup>1</sup> Jour. Amer. Med. Assn., 1913, lxi, 99.

<sup>2</sup> Trans. Amer. Climat. Assn., 1908, xiv, 202.

not in a position to state with any degree of certainty its importance as a factor in the spread of the disease.

**Infection by Inhalation of the Bacilli.**—That the virus of tuberculosis is inhaled with the inspired air has been asserted for centuries by physicians, and Villemin suggested this mode of infection after his experimental investigations. But Koch and his pupil Cornet<sup>1</sup> were the first to prove that dust containing tubercle bacilli derived from dessicated sputum is highly infectious to guinea-pigs. Cornet's experiments with dried sputum scattered over a carpet on which the animals were compelled to live while the carpet was often swept with a stiff broom, has remained classical and is often quoted as proving conclusively the dangers lurking in dried sputum in the average dwelling inhabited by careless consumptives. On the basis of such experiments rested the entire inhalation hypothesis of tuberculous infection.

The fact that diffuse daylight, especially sun-rays, kills tubercle bacilli and soon renders them avirulent would largely exclude infection through sputum deposited in the street and even in large, bright sunny rooms. But the average consumptive, derived as he is from the poorer strata of population, and living in a squalid dwelling lacking sufficient light, may deposit sputum which retains its virulence for a long time.

Many valid objections have been raised against the theory that dessicated tuberculous sputum is the main source of infection in man. Flügge<sup>2</sup> and many others have shown that in the ordinary course of human events things are not as simple as stated by Cornet and Koch. The experiments with the carpet are not altogether analogous to the conditions found in human dwellings, and by no means prove that infection is acquired mainly through the inhalation of dust laden with dried tuberculous sputum. Such large quantities of sputum as were used by Cornet in his experiments on guinea-pigs are exceedingly rarely, if ever, found in the most squalid of dwellings. It is also doubtful whether dust laden with virulent tubercle bacilli is often raised to the height of the human head to be inhaled in sufficient amount to infect even while the floor is being swept.

In fact, further investigations by Flügge, Neisser, Kohlisch, and others have not yielded the same results as those reported by Koch, Cornet and their followers. It was found that in houses inhabited by consumptives the sputum deposited on the floors is not often perfectly dried and thinly pulverized, capable of rising with the dust to the height of five or more feet from the ground. Moreover, conditions in unsanitary homes are not conducive in the direction of drying the sputum soon after it has been eliminated by the consumptive. And if it takes time to dry, it must be remembered that the bacilli lose their virulence within ten days owing to putrefactive processes

<sup>1</sup> Verhandl. Berlin. med. Gesellschaft., 1899, xxx, 91.

<sup>2</sup> Zeitschr. f. Hygiene u. Infektionskr., 1909, xxx, 107.

on the floors of filthy houses, and the diffuse light which acts during the day, or artificially during the night. It is also noteworthy in this connection that in the average house there are no air currents strong enough to raise the dust to the height of about five feet.

It may seem incredible, yet it is a fact that it is exceedingly rare to find a house where proper precautions are taken as to expectoration in which the collected dust shows virulent tubercle bacilli. Even in houses inhabited by consumptives—sanatoriums, dispensaries, railroad stations, factories, cars, etc.—no dust containing virulent tubercle bacilli has been found in most cases investigated. Thus, Kohlisch<sup>1</sup> could not infect guinea-pigs, which are very susceptible, with dust collected in houses inhabited by consumptives; Wagner collected dust in a sanatorium at Zurich, in such places in which the air stream could have brought it, and injected it intraperitoneally into guinea-pigs and found that in only 3.5 per cent. of cases did infection take place. Even in Chaussé's<sup>2</sup> investigations of the dust in the tuberculosis wards of the Hospital Boucicaut in Paris, where conditions are such as to favor bacterial life, only seven out of eighteen specimens showed the presence of virulent bacilli. Dust collected in the streets hardly ever shows the presence of living tubercle bacilli.

**Infection under "Natural" Conditions.**—In a review of the literature on this subject, Charles V. Chapin<sup>3</sup> says: "Although there has been a vast amount of experimental work on infection in tuberculosis, there has been very little in which conditions at all approached the natural. Usually there is an excessive amount of exposure, or an excessive number of germs in spray or dust. Thus, in Cornet's notable experiment, where 47 of 48 guinea-pigs were infected by breathing dust, the carpet has been smeared with large quantities of sputum, and it was forcibly beaten so that clouds of dust rose up directly in front of the animal. It is surprising that so few have thought it worth while to see how infection takes place in animals kept under conditions as nearly as possible like those under which human beings live."

At Dr. Chapin's suggestion, M. S. Packard carried out an experiment with a view of determining the mode of infection under "natural conditions." Two sets of guinea-pigs were exposed in a house occupied by a careless consumptive. They were exposed in cages, one set fed by the patient and the other excluded from any possible form of contact. Most of the animals in both sets developed tuberculosis. Chapin suggests that the animals kept in the locked cage covered with wire gauze were infected by mouth spray, as the patient often held his face right in front of the box and talked to the animals.

Other experiments along these lines were performed by Schroeder and Cotton.<sup>4</sup> They exposed 7 cows in adjoining stalls to 3 tuberculous

<sup>1</sup> *Zeitschr. f. Hyg. u. Infektionskr.*, 1908, lx, 508.

<sup>2</sup> *Ann. Inst. Pasteur*, 1914, xxviii, 720, 771.

<sup>3</sup> *The Sources and Modes of Infection*, p. 309.

<sup>4</sup> *Report Bureau of Animal Industry*, 1906, xxiii, 31.

cows, and found that 6 contracted the disease. They also exposed 100 guinea-pigs in the stalls, 50 in cages below the mangers where food could sift through from the mangers, and 50 on the walls. They also exposed 35 guinea-pigs for one hundred and thirty-five days on the walls of the stalls. Only 2 developed tuberculosis. Of 42 animals kept for fifty-one days under the manger of infected cows, 6 developed tuberculosis of an acute and general type.

There are some points to be borne in mind when evaluating the bearings of these experiments on spontaneous human infection. Guinea-pigs and cattle are more susceptible to infection with tubercle bacilli than are humans, and after all the experiments were not altogether in conformity with conditions in human dwellings. Even Bartel and Spieler's<sup>1</sup> and other attempts to simulate conditions in human contact of tuberculous with non-tuberculous fail when critically examined. It is also a fact that cattle, guinea-pigs, and other animals are "virgin soil," while human beings above the age of fifteen have mostly been immunized by a previous mild infection. Virgin soil is easily infected, as was repeatedly shown. There are many cases on record in which cows in whom the only manifestation of tuberculous infection was that they were "reactors," and were introduced into stables with other cows which did not react to tuberculin. The latter were soon infected, becoming "reactors," though as far as could be ascertained the first infected cows did not excrete any tubercle bacilli. This would indicate that infection can be accomplished in some manner with which we are as yet unacquainted.

**Droplet Infection.**—It is obvious that though infection through the inhalation of dust containing desiccated tuberculous sputum is undoubtedly possible, this is not the only or the most common mode of spontaneous infection of human beings under "natural conditions," and many have maintained that in the vast majority of cases infection is accomplished directly from one person to another. The moist droplets eliminated by consumptives while speaking and especially while coughing and sneezing, may be inhaled by persons who happen to be in their proximity. Flügge<sup>2</sup> and his followers, who have done considerable experimental work along these lines, are satisfied that under natural conditions the dissemination of tuberculosis from man to man, "droplet infection," is the most common mode.

Careful research has shown that the air exhaled by consumptives during ordinary and quiet breathing is free from tubercle bacilli, but the moist droplets eliminated from the mouth while talking, coughing, sneezing, etc., do often contain tubercle bacilli which may remain floating in the air for some time. Indeed, it has been found that the *Bacillus prodigiosus* may thus float in the air for five hours. But this will hardly hold for the tubercle bacillus. After holding a

<sup>1</sup> Wiener klinische Wchnschr., 1905, xviii, 218.

<sup>2</sup> Die Verbreitungsweise und Bekämpfung der Tuberkulose auf Grund experimenteller Untersuchungen, Leipzig, 1908.

cover-glass in front of a coughing consumptive, tubercle bacilli were found microscopically as well as by inoculation experiments which were positive in 90 per cent. of cases. In many cases bacilli were deposited on the cover-glasses, which were held at a distance of from 40 to 80 cm. from the patient's mouth. The infectiousness of these droplets was confirmed by experiments of Heymann<sup>1</sup> who exposed guinea-pigs in front of coughing consumptives.

These experiments were apparently more often positive than in the case of infection with dust containing desiccated tuberculous sputum, and Flügge and his followers conclude that this mode of infection is the most important under natural conditions.

But even these experiments are open to question. The animals were held tightly for hours directly exposed to the faces of the consumptives who coughed directly into their open mouths. Such exposure never occurs in human beings, except perhaps in cases of tuberculous mothers holding their crying babies on their arms and coughing directly into their open mouths, which may be observed now and then among certain classes, but after all cannot be considered very common.

Even conceding that droplet infection is an important mode of transmission of tuberculosis, it must be realized that it depends on many factors which are not always, nor even often, operative. It has been found that when a healthy person is at a distance of three feet from the coughing patient, the droplets will not reach far enough to become a possible infective agent excepting perhaps when carried by air currents. Another important factor is the dose of the bacilli that may thus be inhaled. It has been shown that small numbers of bacilli are easily taken care of by the human organism. It is also a fact that tubercle bacilli thus eliminated do not remain floating in the air for any length of time, but sink to the floor where they are soon rendered innocuous, as was already mentioned.

It is thus obvious that only when contact with the consumptive is very close, intimate, and prolonged, which in ordinary life occurs, as a rule, only in mothers with sucking infants, or between husband and wife, droplet infection may become a serious menace. And even in these cases there are natural safeguards.

Considering the evidence thus far brought together at its face value, it appears that inhalation of dust containing tuberculous sputum, or of droplets expelled by consumptives while talking, coughing, and sneezing, may infect a healthy person, yet the evidence that these are the most frequent modes of the dissemination of tuberculosis is inadequate. Behring and Chapin have aptly pointed out that there has been a strong tendency among medical men of all ages to attribute the transmission of infectious diseases of obscure causation to the inhalation of the virus. This has notably been the case with the endemic

<sup>1</sup> Quoted from Flügge.



diseases of childhood, and for a long time yellow fever, typhoid, typhus, malaria, relapsing fever, etc., were all considered inhalation diseases and proofs were at hand to substantiate these contentions. Recently more exact studies have shown conclusively in some, and with a high degree of probability in others, that they are altogether transmitted through the agency of certain insects. Indeed, physicians of a few generations ago drew analogies between tuberculosis and malaria, typhus, etc., showing that they were all caused by the inhalation of the virus.

**Natural Barriers against Inhalation Infection.**—Notwithstanding the various disharmonies which may be found in the structure and functions of the human body, and which Metchnikoff has so cleverly enumerated in one of his books, the respiratory tract is provided with a most wonderful protective apparatus for the prevention of the entry and implantation of bacilli in the deeper respiratory passages. Indeed, no organ in the body, excepting the central nervous system, is fitted out with better safeguards in this regard.

The bacilli cannot enter the lungs with ease. The nasal passages, mouth and throat act as excellent filters, detaining the inhaled dust. Even when some microorganisms in the inhaled air pass all the barriers, the mucus secreted all along the tract, the ciliated epithelium, etc., soon remove them as foreign bodies when necessary, assisted by cough, which has the function of clearing the lungs. The few bacilli which may remain within for any reason are under normal conditions well cared for by the extensive lymphatic apparatus which surrounds all the bronchi and bloodvessels and takes up bacteria, destroying them or at least rendering them innocuous. From animal experiments conducted for years by Bacmeister<sup>1</sup> he shows that while tubercle bacilli are only rarely found in the lungs of animals compelled to inhale dust containing the germs, he never observed that infection of the normal lung should be caused in this manner, and he concludes that the bacilli must be hindered in their development, destroyed or carried away from the lungs by the lymph and blood stream. There is no reason against the assumption that the normal human lung acts in the same manner and that small numbers of bacilli which may succeed in penetrating into deep air vesicles are removed or destroyed before they can gain a foothold and cause disease.

It must, however, be borne in mind that dust of any kind may and does reach the lungs with the inspired air, as is evident from the large number of cases of pneumokoniosis of various degrees. Tubercle bacilli may thus be brought there with the inspired air. But whether they cause disease in every case they reach the lungs is a disputed problem, the weight of evidence being against such a contention. Indeed it has been proved that tubercle bacilli may remain alive and virulent in the tracheobronchial glands for years without causing

<sup>1</sup> Die Entstehung der menschlichen Lungenphthise, Berlin, 1914.

disease, or even changes in the glands. Investigations by Bartel and Weichselbaum, Harbitz and others have shown that this is frequently the case, and it explains the latency of tuberculosis in many cases.

That tubercle bacilli on mucous membranes are not invariably causing disease is proved by another fact. These microorganisms have been found on the mucous membranes of the nose, throat and mouth of healthy individuals. Noble W. Jones<sup>1</sup> found them in the nasal cavities of healthy persons in the ordinary walks of life, especially those who cared for consumptive patients. Strauss<sup>2</sup> found tubercle bacilli in the nasal cavities of healthy individuals living in houses inhabited by phthisical patients. Alexander<sup>3</sup> found them in very large numbers on the mucous membranes of patients suffering from ozena, but who had no symptoms or signs of tuberculosis. These facts, taken in connection with the fact that tuberculosis of mucous membranes of the pharynx, nose and mouth is exceedingly rare even in consumptives, show that these structures possess a certain natural resistance against tuberculosis. That it is not solely due to the immunity acquired by previous tuberculous infection is shown by the fact that, as a primary infection, tuberculosis of these parts is exceedingly rare, though it must be admitted that while entering the body, by inhalation or ingestion, the bacilli must pass them.

A lymphatic apparatus of normal structure and function evidently insures against the implantation of all kinds of bacilli in the respiratory passages. Otherwise we would all succumb to various diseases, including tuberculosis. It is only when the natural protective forces fail that tuberculous infection may be caused in this manner.

On the other hand, it must be emphasized that the lungs are very much exposed to infection from the blood stream, and hematogenic infection may easily localize itself in these organs. The lungs are the first filter for everything that may be carried by the venous circulation. When the lymphatic apparatus is injured by anthracosis, which is very frequent in city dwellers, it is not capable of removing tubercle bacilli which may be brought to them with the blood stream. The apices are also located in an especially unfavorable position and do not move with the respiratory activity as well as the lower parts, and when to this are added an ossified costal cartilage and a short first rib, we have everything favorable for the localization of bacilli in the apices (see Chapter IV).

**Difficulties in the Way of Establishing the Portals of Entry of Tubercle Bacilli.**—The reasons why experimental investigations have failed to adequately solve the problems of the aërogenic etiology of phthisis are evident when we bear in mind that pulmonary tuberculosis, as met with in human beings, showing isolated foci which extend

<sup>1</sup> Med. Record, 1900, lviii, 285.

<sup>2</sup> Bull. de l'Acad. de Méd., Paris, 1894, xxxii, ii, 18.

<sup>3</sup> Berl. klin. Wehnschr., 1903, xl, 508.

slowly downward in the lungs, never occurs spontaneously in animals; nor has it ever been induced artificially or experimentally in animals.

Real active initial lesions in the human lungs have only rarely been encountered at necropsies. Most cases examined on the autopsy table are advanced and it is very difficult, or impossible, to decide which was the initial lesion. Even the few initial lesions, found in cases that have died from causes other than tuberculosis, and reported by Schmorl,<sup>1</sup> Birch-Hirschfeld,<sup>2</sup> Lubarsch,<sup>3</sup> Beitzke,<sup>4</sup> and others have not cleared up definitely the problem whether the bacilli were brought to the site of the lesion by the inspired air or the blood stream. It has, however, been found that even at that stage both the bronchioles and the bloodvessels were affected to such an extent that either or both could be considered the portal of entry.

The fact that the regional lymphatic glands and lymph nodes are usually implicated at an early stage points to a hematogenic localization, but it may also be explained by the aërogenic hypothesis.

It is obvious that the inhalation of the bacilli does not exclude hematogenic distribution and their final localization at some point distant from the point of entry. Ribbert, Bacmeister, Lubarsch and others have pointed out that microorganisms brought into the bronchial tree by the inspired air may pass through the mucous membrane into the lung tissue without producing a visible lesion at the point of entry; pass along the lymphatics into the regional lymph nodes and from there carried by the blood stream into the pulmonary apices. But that this is in all probabilities rare, may be assumed when it is recalled that only few bacilli can reach the bronchi, and of these but few are allowed to pass through the normal mucous membrane of these tubes and the alveoli, and they are usually rendered innocuous by the protective properties and functions of the lymph and blood, as was just shown.

**Hematogenic Infection.**—Many look at phthisis as hematogenic in origin: The tubercle bacilli are assumed to enter the body at any point, the respiratory or digestive tract, or even through the skin, and are carried by the blood stream until they reach a point where the tissues have a low power of resistance, an organ which offers a favorable soil for the growth and action of these microorganisms. Considering the enormous frequency of pulmonary phthisis, it is evident that in the vast majority of human beings the lungs offer a good breeding-point for the tubercle bacilli. The localization of the bacilli is thus accomplished in the same manner as their localization in joints, the peritoneum, the meninges, etc.—by the blood stream.

The hematogenic origin of phthisis is especially urged by Baumgarten, Ribbert, and Aufrecht. According to Baumgarten, tubercle

<sup>1</sup> München. mediz. Wehnschr., 1902, xlix, 1379.

<sup>2</sup> Deutsches Arch. f. klin. Med., 1899, lxiv, 58.

<sup>3</sup> Virchows Archiv, 1913, cexiii.

<sup>4</sup> Berl. klin. Wehnschr., 1909, xlvi, 388.

bacilli in the inspired air may infect the mucous membranes of the upper respiratory tract whence they are carried by the lymphatics to the regional glands—the submaxillary, cervical, and supraclavicular, which are so often enlarged in tuberculous children. Entering the superior vena cava they may be carried by the blood stream to the lungs, causing typical interstitial tubercle of these organs and finally extend, while growing, to the alveolar walls or within them. Aufrecht holds that the primary tuberculous lesion is always in the vascular walls, which are affected by bacilli brought to them by the blood stream. Through the veins they pass into the right heart; or from tuberculous bronchial glands they get into the pulmonary artery or its branches, when the lymph channels are obliterated by inflammatory processes, into the finest bloodvessels and capillaries. Aufrecht has quite some experimental work in support of his contention.

It is thus clear that the aërogenic hypothesis of the origin of phthisis is explained by either a *hematogenic* or *lymphogenic* localization of the bacilli in the lungs. The frequency of tuberculosis of the glands, serous surfaces and meninges speaks in favor of such origin of lung disease. The recent discoveries to the effect that a bacteremia is very frequent in phthisis support this contention.

**Infection by Ingestion.**—The most important mode of hematogenic infection in phthisis should be the ingestion of tubercle bacilli, although it by no means excludes the air passages as portals of entry, because germs inhaled through the mouth, nose, and throat may be swallowed and pass into the blood through the mucous membranes at any point of the gastro-intestinal tract. However, in the vast majority of cases it would be with food, especially with milk from tuberculous cows, that the bacilli would enter the body and cause disease.

Simple as this theory appears, there are many objections to be considered before accepting it. The assertions of some authors that tubercle bacilli are invariably killed by the gastro-intestinal juices has been found largely incorrect, as was pointed out by Römer. To be sure, the gastro-intestinal juices may, and usually do, interfere with their rapid proliferation, and so may any fermentation in the intestinal tract, while the peristaltic movements of the intestines may soon remove them from the body; but they are not necessarily killed. Moreover, while a healthy, unbroken mucous membrane of the digestive tract is impermeable to tubercle bacilli, it is clear that a perfectly normal mucous membrane is very rare considering the different kinds of food and its débris which pass through it, and the least disturbance in its anatomical structure or function may be sufficient to permit the passage of bacteria through its walls.

Experimental investigations have shown that feeding guinea-pigs, rabbits, and monkeys with tuberculous sputum, or with pure cultures of tubercle bacilli is effective in infecting the animal. Moreover, it has been found that the bacilli may pass through the intestinal walls into the blood or lymphatics without leaving any trace on the walls

of the canal. In some experiments it was found that a couple of hours after the ingestion the bacilli were already in the chyle.

Because of this possibility of the tubercle bacilli entering the blood or lymph stream from the digestive tract, various authors have suggested the different parts of the canal from the mouth to the rectum as portals or entry of the bacilli, which are taken up by the blood and carried to the lungs where they finally stay and cause phthisis. Some have stated that irritated gums during dentition of infants offer a good portal of entry for the bacilli; the frequency of enlarged cervical glands at that period of life was cited as a good proof of the theory. Others have accused the tonsils, especially the pharyngeal tonsil. From the regional cervical glands some authors have traced the bacilli to the bronchial glands and finally to the lungs, though this has been shown by Wood<sup>1</sup> and Beitzke<sup>2</sup> not feasible for anatomical reasons. However, it must be acknowledged that even if there is no anatomical connection favoring the migration of bacilli from the cervical glands to the lungs, the microorganisms may be carried to any place by the blood. On the other hand, it must be mentioned that the tracheo-bronchial glands may be infected directly from the lungs by bacilli which have reached them with the inspired air.

The most conclusive proof of the tubercle bacilli entering the lungs *via* the digestive tract has been brought forward by Calmette and his school, also by Ravenel, Whitla, and many others. Calmette<sup>3</sup> denies dust containing tubercle bacilli as a strong factor in phthisiogenesis. He could not produce anthracosis in animals after subjecting them to prolonged inhalation of air saturated with lampblack. Introducing dry or moist tubercle bacilli directly into the trachea by inhalation or insufflation, or even by inoculation, they were never found to reach farther than the bifurcation of the trachea. Introducing lampblack into the stomach through a tube, thus excluding inhalation, or mixing it with food, anthracosis was soon produced in the lungs of the animals. Similarly, tubercle bacilli introduced carefully into the stomach through a tube with a view of preventing aspiration into the trachea, invariably produced tuberculosis. Ravenel,<sup>4</sup>Schlossmann, and St. Engel could recover tubercle bacilli from the lungs within a few hours after placing them in the stomach by celiotomy.

Sir William Whitla's<sup>5</sup> experiments along these lines are very instructive. He injected a mixture of China ink and water into the large vein in the ear of a rabbit. The animal was killed an hour later and its lungs were found highly charged with carbon particles. He fed for four days a guinea-pig with an emulsion made by rubbing up finely powdered China ink in olive oil and water. The lung was found

<sup>1</sup> Ann. Rep. Henry Phipps Inst., 1906, iv, 163.

<sup>2</sup> Virchows Archiv, 1906, clxxxiv, 1.

<sup>3</sup> Ann. de l'inst. Pasteur, 1905, xix, 601; 1906, xx, 353.

<sup>4</sup> Cleveland Med. Jour., 1909, viii, 179.

<sup>5</sup> Lancet, 1908, ii, 135.

blackened by disseminated particles of carbon in the upper and along the margins of the lower lobes within from eight to twenty-four hours after a single dose. Whitla thus explains the migration of the carbon from the gastro-intestinal tract to the lungs: The carbon particles effect an easy entrance through the intestinal epithelial surface; reaching the lacteal or lymphatic paths they pass through the lymphatic glands of the mesentery, and finally, either inclosed in phagocytes or free, find their way into the thoracic duct to be poured into the venous circulation before being arrested in the capillaries of the lungs. Vasteenburgh and Grysez's experiments have also shown that it is easy to render an adult guinea-pig perfectly anthracotic without subjecting it to repeated inhalations of carbon particles.

Considerable work along these lines has been done in this country. Schroeder and Cotton<sup>1</sup> found that no matter in what part of the body tubercle bacilli are inoculated, pulmonary disease may result. Charles V. Chapin,<sup>2</sup> reviewing the literature on the subject says that "the unprejudiced reader must conclude that infection in either way is possible (inhalation and ingestion), but the conditions of the experiments are so far removed from the natural that there must be much hesitation before assuming that this work indicates in any degree the common mode of infection in human beings."

From these and many other experiments we are safe in concluding that tuberculous infection, including phthisis, may be acquired through the ingestion of tubercle bacilli, and that the digestive tract permits the passage of the bacilli, which are carried by the blood and lymph streams to the various points of least resistance, of which, in the human being, the pulmonary apices appear to be the most vulnerable.

We must not, however, overlook the fact which has been established experimentally, that large numbers of bacilli are necessary to accomplish results and the normal gastro-intestinal tract can easily dispose of small doses of tubercle bacilli.

Ingestion of tubercle bacilli may result in tuberculosis of the cervical or mesenteric glands, depending on the point at which the bacilli enter the upper or lower parts of the canal. From these glands the bacilli are taken up by the circulating blood and carried to the tracheo-bronchial or mesenteric glands and to the lungs. In many cases the bacilli remain dormant in these glands indefinitely, causing no disease at all; in others the latency lasts only for some time, when finally, because of some exciting cause, they flare up again, migrate with the blood stream and, localizing in the lung, cause phthisis, and we then think that we are dealing with a new infection.

Autopsies made by Gaffky,<sup>3</sup> Ungermann, Wollstein and Bartlett,<sup>4</sup> Ghon,<sup>5</sup> Hamburger,<sup>6</sup> and others have shown that in children both

<sup>1</sup> Rep. Bur. Anim. Industry, 1906, xxiii, 31.

<sup>2</sup> The Sources and Modes of Infection, New York, 1912, p. 301.

<sup>3</sup> Tuberkulosis, 1907, vi, 437.

<sup>4</sup> Amer. Jour. Child. Dis., 1914, viii, 362.

<sup>5</sup> Der primäre Lungenherd bei der Tuberkulose der Kinder, Berlin, 1912.

<sup>6</sup> Die Tuberkulose des Kindesalter, Vienna, 1912.

glandular systems—the abdominal and the thoracic—are affected in nearly the same proportion. Primary infection of the intestine is very rare in adults, though in children it is quite common. Behring, however, believes that all infections date back to early infancy when the bacilli were ingested, remained latent to flare up again in later years, causing disease of the lungs (see Chapter V). Of course while making autopsies on adults who died from chronic tuberculosis it is difficult or impossible to find the point of primary inoculation. But in infants and children this may be done in most cases. Perhaps one of the best criteria is that in primary intestinal infection the mesenteric glands are implicated while the intestinal mucous membrane may remain intact, and in secondary intestinal tuberculosis—the ulcerations so frequently found in phthisical subjects—the mesenteric glands are only rarely affected. Statistics of primary tuberculosis of the intestine in children are not in accord. From the data published by Orth, Eden, Councilman, Mallory and Pearce, Lubarsch, Wollstein and Bartlett and many others, it appears that the percentage ranges from five to fifty. A large proportion of these infections are due to bovine bacilli, as was already shown (p. 28).

**Significance of Bovine Infection.**—Nor can we decide upon the channels of entry of the tubercle bacilli by a study of the type of these microorganisms found in the case. We must bear in mind that cow's milk contains tubercle bacilli more frequently than has been appreciated. The studies of E. C. Schroeder,<sup>1</sup> John F. Anderson,<sup>2</sup> Ravenel and others have shown this to be a fact in this country. M. Rosenau<sup>3</sup> compiled data concerning 551 samples of milk examined in which tubercle bacilli were found in 46, or 8.3 per cent., and he says that this may be taken as the average percentage for the entire country. But practically all the cases of pulmonary phthisis are due to the human type of bacilli, and in countries where milk is hardly used as a food, as is the case in Japan, China, India, Egypt, etc., phthisis is not lacking, as has been shown by Kitasato<sup>4</sup> and others. Moreover, the Imperial Health Department of Germany has made a collective investigation on the subject of bovine infection as a cause of tuberculosis of the lungs and found that out of 280 children, all of whom had been fed since infancy on milk derived from cows with tuberculous udders, only 2 became sick with tuberculosis during seven years, and not a single case of death occurred among them.

We have seen that the tuberculosis in children caused by bovine infection consists almost invariably in diseased glands, skin, bones, joints and intestines, and fatal phthisis is exceedingly rare (p. 29). There is also ample evidence that the adult is practically immune to the bovine bacilli, even if his immunity to the human type of bacilli

<sup>1</sup> Bull. No. 99, Bureau of Animal Industry, 1907.

<sup>2</sup> Jour. Infect. Dis., 1908, v, 107.

<sup>3</sup> Preventive Medicine, New York, 1913, p. 513.

<sup>4</sup> Sixth Intern. Congr. on Tuberculosis, 1908, vi, 1.

has not yet been established to the satisfaction of all.<sup>1</sup> Younger individuals, when infected with the bovine type of bacilli find it more or less easy to cope with the situation and to recover, even if they finally emerge with disfigurement, or perhaps crippled. But if the problem of tuberculosis was only that part which is produced by the bovine bacilli, it would not have by far the significance it has at present. In fact several authors, especially Riviere,<sup>2</sup> are of the opinion that these mild bovine infections immunize the organisms against infection with the more virulent human type, as will be discussed later on.

**Evaluation of Experimental Data.**—On the whole the experimental evidence, though ample in quantity, is not always in agreement with what would be expected *a priori*; nor are the results of one investigation invariably the same as those obtained by another who ostensibly followed the same method.

The differences in the results of experimental investigations are best explained by lack of equilibrium between the host and the parasite, as has been found by many bacteriologists, notably Theobald Smith,<sup>3</sup> who says: "It varies with the species, race, nationality, or even family of the host and many other accessory conditions. It depends on the race of the tubercle bacilli. In experiments such conditions as age of culture, total period of cultivation, character of the culture medium, condition of aggregation of the bacilli, mode of application and dosage are of great importance in determining the outcome of the experiment. Similarly, the outcome will vary according to the species of animal on which we are experimenting."

There are other reasons why we should be careful before applying experimental finding to clinical medicine. Even among various species of animals the results are not always the same when an experiment has been performed in the same identical manner and with the same culture of bacilli. Thus, as has been pointed out by Weber, after inoculating subcutaneously guinea-pigs with bovine bacilli there results

<sup>1</sup> Inasmuch as this may appear to be a sweeping statement I will cite at some detail Felix Klemperer's experiments: In February, 1900, he injected subcutaneously bovine bacilli into his arm. Ten months later he excised the indurated subcutaneous cellular tissue at the site of the injection. Microscopic examination showed well-organized granulation tissue with giant cells but no caseation. No tubercle bacilli could be discovered, showing that tuberculosis was probably absent, and the tissue changes were at any rate not characteristic of tuberculosis. Another physician, who had been tuberculous for fourteen years, also submitted to similar injections of bovine bacilli. In this experiment the individual was given fourteen injections without producing any results. Four other tuberculous patients were injected with tuberculous lymphatic tissue from guinea-pigs. A total number of thirty-nine injections of bovine bacilli were administered to these four patients. The local effects were slight. Four times abscesses were produced which, however, healed sooner or later. General constitutional effects were not observed in any case, the patients even stated that they felt better and they gained in weight during the treatment. Klemperer concludes that there is no doubt that subcutaneous injection of bovine bacilli is, within certain limits, harmless to the tuberculous individual (*Zeitschr. f. klin. Med.*, 1905, lvi, 241). Baumgarten performed similar experiments on cancerous patients with the same results.

<sup>2</sup> *British Journal of Tuberculosis*, 1914, viii, 83.

<sup>3</sup> *Harvey Lectures*, 1905-1906, p. 273.



disease first of the spleen, and second of the liver, but the kidneys are almost never affected, while in the rabbit the kidneys are affected next to the lungs. The lung of the hen is practically refractory to the *typus gallinaceus* of the acid-fast bacilli. In the rabbit there is always an infection of the lymph glands after inoculation of bovine bacilli, but when human bacilli are inoculated these glands are never affected. Inasmuch as the internal organs are affected after subcutaneous inoculation, it is evident that the bacilli pass the regional lymph glands without harming them. Rats respond to infection with the human type of bacilli in the same way as rabbits. There is no doubt that various strains of the same type of bacillus produce different results when inoculated into the same species of animals, and in humans the different types of disease resulting from infection may undoubtedly be attributed to the same causes. Chronic phthisis is a distinctly human disease which never occurs in animals spontaneously, nor has it ever been induced experimentally.

**Conclusions.**—A survey of the evidence presented in this chapter shows clearly that there is no agreement among authorities as to the most common channel of entry of the tubercle bacilli before causing phthisis. The reason is clear when we bear in mind that experimental investigations in laboratories have in most cases not duplicated natural conditions among human beings. Charles V. Chapin, who has so well pointed out this fact, arrives at the conclusion that it is highly desirable that a sufficient number of well-conducted experiments under truly natural conditions be made to determine the relative importance of inhalation of desiccated sputum, and the ingestion of the bacilli in the spread of the disease. Römer, perhaps the most indefatigable experimental worker in the field of tuberculosis, also says that there is evidently some mode of transmission of this disease with which we are as yet unacquainted.

It must, however, be mentioned here, a point which will be discussed in detail later on, that infection alone is not sufficient to produce phthisis; the disease occurs after all in only a certain proportion of persons infected with the tubercle bacilli. In other words, while there is no phthisis without tubercle bacilli, these microorganisms can only harm one who is predisposed to the disease. Under the circumstances phthisiogenesis is more a problem of predisposition than of infection.

## CHAPTER III.

### THE EPIDEMIOLOGY OF TUBERCULOSIS.

**Ubiquity of the Tubercle Bacillus.**—In our survey of the biological characteristics and the channels of entry of the tubercle bacilli we found that the virus of tuberculosis is ubiquitous; that it is found wherever civilized human beings congregate, because tuberculous human beings expectorate sputum containing these bacilli, and domestic animals affected with this disease are everywhere. It has been estimated that the number of bacilli discharged daily in the sputum of a single patient with advanced phthisis is as great as the number of human beings on the earth. The modest estimate mentioned by Cornet may be taken as near the truth—that 7,200,000,000 bacilli may be thrown off daily from a single patient. If we imagine each organism placed end to end in a single file, this number would constitute a chain no less than twelve miles in length.

Clinical and experimental medicine have shown conclusively that the expectoration of consumptives, milk from tuberculous animals, etc., are capable of causing infection; that these microorganisms may enter the body through wounds, as well as through the unbroken skin, and the mucous surfaces of the respiratory and alimentary tracts, etc. We have also shown that though there are many hindrances in the way of infection, still when everything stated in the last chapter is considered, it is not surprising that one out of eight in civilized countries succumbs to the disease, but that the other seven escape its ravages.

**Tuberculous Infection vs. Tuberculous Disease.**—As a matter of fact very few escape infection with the tubercle bacilli, especially of those living in large industrial cities. When we make this statement we want to emphasize that a distinction is to be made between tuberculous infection and tuberculous disease. The latter refers to the disease known for centuries, ever since Hippocrates described it, as consumption or the equivalent of the term found in all European languages. It is the disease which causes more than 95 per cent. of the suffering, social and economic misery and deaths due to the tubercle bacilli. On the other hand, tuberculous infection covers all the cases in which the virus of tuberculosis has entered the body, irrespective whether it has caused disease or not. Tuberculous disease is always preceded by infection, but infection with the tubercle bacilli is not invariably followed by disease.

Research of the past three decades has shown conclusively that

infection with tubercle bacilli is not invariably followed by that train of symptoms which we observe in phthisis; that it does not necessarily cause any sickness, excepting an altered reactivity to tuberculin. Apparently more people harbor the bacilli within their bodies, or show traces of having harbored them, without knowing it at all, than such as suffer or succumb as a result of tuberculosis of the lungs or other organs. These persons are undoubtedly tuberculous, and there are many strong reasons that, like other bacillus "carriers," they are liable to cause mild infection with tuberculosis. But they are not at all phthisical in the clinical sense. Some of them are destined to become phthisical; in fact practically all phthisis evolves from an infection acquired during childhood, as we shall show when discussing phthisiogenesis.

**Frequency of Tuberculous Infection.**—Careful and painstaking scientific investigations have shown that the frequency of tuberculous infection goes hand in hand with civilization, or contact of primitive peoples with civilized humanity. In modern large cities very few persons escape infection. Autopsies made with a view of ascertaining traces of tuberculous lesions, both active and healed, have shown that over 90 per cent. of adults are thus affected among the civilized; but among primitive peoples no tuberculous changes are found at autopsies.

In Laennec's<sup>1</sup> classical work on diseases of the lungs published in 1831 we find the following in a footnote: "M. Lombard's investigations in the Children's Hospital at Paris show that of the children who die between one and two years of age, one-eighth are tuberculous; between two and three, two-sevenths; between three and four, four-sevenths; between four and five, three-fourths. In the succeeding years up to puberty, tubercles are found more frequently than before the fourth, but much less frequently than from the fourth to the fifth. Papavoine, of the same hospital, found that the number of tuberculous children between the fourth and eleventh years is greater than those who are not tuberculous, tubercles being particularly prevalent from the fourth to the seventh years. Their frequency is again increased about the twelfth and thirteenth years, and at fourteen and fifteen years the rate of prevalence is the same as at four and five. These results were obtained from investigations made on 910 children (388 boys and 522 girls); somewhat less than three-fifths were tuberculous."

Similarly, Henry Ancell<sup>2</sup> emphasized the extent of tuberculous disease in London as far back as 1840. In a paper on "Facts and Opinions Relating to Tuberculosis, with Commentaries," he cites the *Decenium Pathologicum* of Dr. L. K. Chambers, giving the results of the post-mortem examinations made in the mortuary of St. George's Hospital

<sup>1</sup> *Traité de l'auscultation médiate et des maladies des poumons et du cœur*, Paris, 1831, ii, 125.

<sup>2</sup> *Association Med. Journal*, 1853, p. 1030; quoted from Karl Pearson, *loc. cit.*, p. 19.

in the ten years, December 31, 1840, to December 31, 1850. The number of autopsies was 2046. The following are the figures:

	Birth to 15 years.	15 to 30.	30 to 45.	45 to 60.	Above 60.	All.
Total number of autopsies . . .	154	636	651	438	167	2046
Per cent. of tubercle found . . .	29.7	35.8	25.8	19.6	7.7	26.1

It appears that these facts were entirely forgotten, and medical literature was silent about the extent of tuberculous infection and changes in the bodies of many who have shown no indication of disease during life, until in 1900 Naegeli<sup>1</sup> published his report of 500 autopsies at the Pathological Institute at Zurich. He found 71 per cent. showed pathological changes due to tuberculosis. Among individuals under eighteen years of age, only 25 per cent. showed such lesions, mostly of a grave character, often leading to a fatal termination. But in persons above eighteen years of age the proportion that showed traces of tuberculous infection reached 98 per cent. Of these only 28 per cent. died as a result of this disease, while the rest had tuberculous foci which were either altogether healed, quiescent, or slowly progressing.

When first published this revelation appeared incredible, but when other pathologists investigated autopsy material along the same lines, they practically confirmed Naegeli's findings. From the works of Harbitz, Scheel, Burckhardt, Lubarsch, Adami<sup>2</sup> and McCrea, and many others, it was clear that very few persons escape infection with tubercle bacilli before reaching the age of maturity. They have all found that no matter what the cause of death may have been, whether the persons knew that they had been tuberculous or not, between 50 and 100 per cent. of people living in large cities show active, quiescent or healed tuberculous lesions in some organs of their bodies. On this point all are now in agreement, the only dispute which may be found in the literature consists in whether the percentage is only 70, or reaches as high as 100. Thus, Lubarsch<sup>3</sup> states that Naegeli has exaggerated his findings, because of 7371 necropsies performed by Naegeli, Burckhardt, Risel and Lubarsch, only 4230, or 57.4 per cent., showed tuberculous changes; of 5796 necropsies on adults, 4017, or 69.2 per cent., showed such changes.

These autopsies showed another significant fact: The newborn infant is invariably free from tuberculosis, indicating that infection, if it occurs at all, always takes place after birth. Among infants dying during the first year of life from any cause, some are found presenting lesions of a tuberculous character, while beginning with the second year the number of infected children increases steadily, so that at the age of fifteen there are nearly as many tuberculous among them as among adults. In this country Martha Wollstein

<sup>1</sup> Virchows Archiv, 1900, clx, 426.

<sup>2</sup> Trans. Sixth Internat. Congr. on Tuberculosis, 1908, i, 325.

<sup>3</sup> Virchows Archiv, 1913, cexiii, 417.

and F. H. Bartlett<sup>1</sup> reported 1320 autopsies performed at the Babies' Hospital in New York City on children under five years of age, of which 118, or 13.5 per cent., showed tuberculous changes. In Europe the proportion is even higher, as is evident from the finding of Naegeli, Burckhardt, Lubarsch, Hamburger, and many others.

The most recent series of autopsies on children has been reported by Harbitz.<sup>2</sup> In the Anatomical Institute at Christiania, Sweden, during 1898 to 1911, the bodies of 484 children who died from any cause were dissected. The ages ranged from birth to fifteen years. His results are given in the following figures:

Age.	Number examined.	Tuberculous lesions. Per cent.
0 to 1 year . . . . .	201	20.0
1 to 2 years . . . . .	65	26.2
3 to 4 " . . . . .	44	31.8
5 to 6 " . . . . .	28	67.9
7 to 10 " . . . . .	53	62.2
11 to 14 " . . . . .	53	81.1
15 " . . . . .	40	80.0
Total . . . . .	484	41.08

The anatomical picture was predominantly that of tuberculosis of the lungs and the lymphatic glands, especially those of the thorax. The younger the child, the more acute and progressive the lesion found. In only one case could he suspect congenital tuberculosis.

Another point has been brought out by these autopsies which is of immense epidemiological and clinical importance. The tuberculous lesions found at the autopsies are not all active, nor were they the cause of death in many cases. Indeed, there were many which were latent, quiescent, or even healed. Thus among the 406 tuberculous bodies examined by Naegeli, 28.1 per cent. had healed or latent lesions; among Burckhardt's 1452 autopsies he found 1221, or 84.1 per cent., tuberculous; but 39.4 per cent. of them showed quiescent, latent or healed lesions. The results of nearly all other investigations show the same conditions.

Active and progressive lesions, leading to death, are characteristic of infancy; in fact, during the first year of life all lesions discovered at autopsies are those of a generalized and progressive tuberculosis. Localized lesions are rare in childhood, and only make their appearance after the second year, but are still rare at ten years of age. Lubarsch has analyzed his findings from this viewpoint and arrives at the conclusion that the younger the individual infected with tuberculosis, the more likely he is to be killed by the disease, while the older the individual, the less is he likely to suffer from acute and progressive disease. In fact, he says that among older persons tuberculosis is a relatively harmless process, showing as it does a strong tendency

<sup>1</sup> Amer. Jour. Dis. Child., 1914, viii, 364.

<sup>2</sup> Norsk mag. f. Laegevidesk., 1913, 5 R., xi, 1.

to latency or healing. He illustrates this point by the following statistical facts:

Among 502 infants under one year examined after death, 4.58 per cent. were found with tuberculous lesions all of which were acute or subacute general tuberculosis, without any tendency to localization in a single organ. Of 123 children two years of age, 20.3 per cent. were found with tuberculous lesions. All were also active and progressive, though there were already seen tendencies to localization of the process, but no calcification was noted. At three years of age 24.7 per cent. of the bodies showed tuberculous changes, and in one some evidences of calcification were found microscopically in a tuberculous bronchial gland. He found that the number of active and fatal cases of tuberculosis keeps up at a high level till the age of fifteen, when localized tuberculosis begins to manifest itself, though the lesions still show tendencies to progression, and calcification is still exceptional. Thus, among 139 tuberculous bodies of individuals between one and sixteen years of age, only 33, or 23.7 per cent. showed calcified foci, but none were completely healed—all were active and progressive in character.

Only after the seventeenth year of life are to be noted latent and healed tuberculous lesions at autopsies, and they keep on increasing in frequency, so that at the age of forty they are more frequent than progressive lesions. The following table, as well as Fig. 1, shows the point clearly:

Age.	Active lesions. Per cent.	Latent and healed lesions. Per cent.
17 to 20 . . . . .	77.4	22.6
20 to 30 . . . . .	76.7	23.3
30 to 40 . . . . .	52.6	47.4
40 to 50 . . . . .	38.9	61.1
50 to 60 . . . . .	33.5	66.5
60 to 70 . . . . .	23.3	76.7
70 to 80 . . . . .	14.7	85.3
80 to 90 . . . . .	9.3	90.7
90 to 100 . . . . .	0.0	100.0

The frightful tuberculization of humanity, as revealed by these autopsy findings, was explained by some authors as due to the fact that in hospitals there is a concentration of tuberculous sick, and among children who succumb at an early age, the percentage of tuberculous should be much higher than among those who survive till maturity. But it must be recalled that these autopsy findings were obtained in children who died from all causes, and that in many the tuberculous lesions were found incidentally, although the causes of death were entirely different diseases.

Many objections have been raised against these autopsy statistics showing that nearly all adults living in cities harbor tuberculous lesions in their bodies. Some have maintained that many non-tuberculous changes in the lungs and pleura have been included as latent or healed

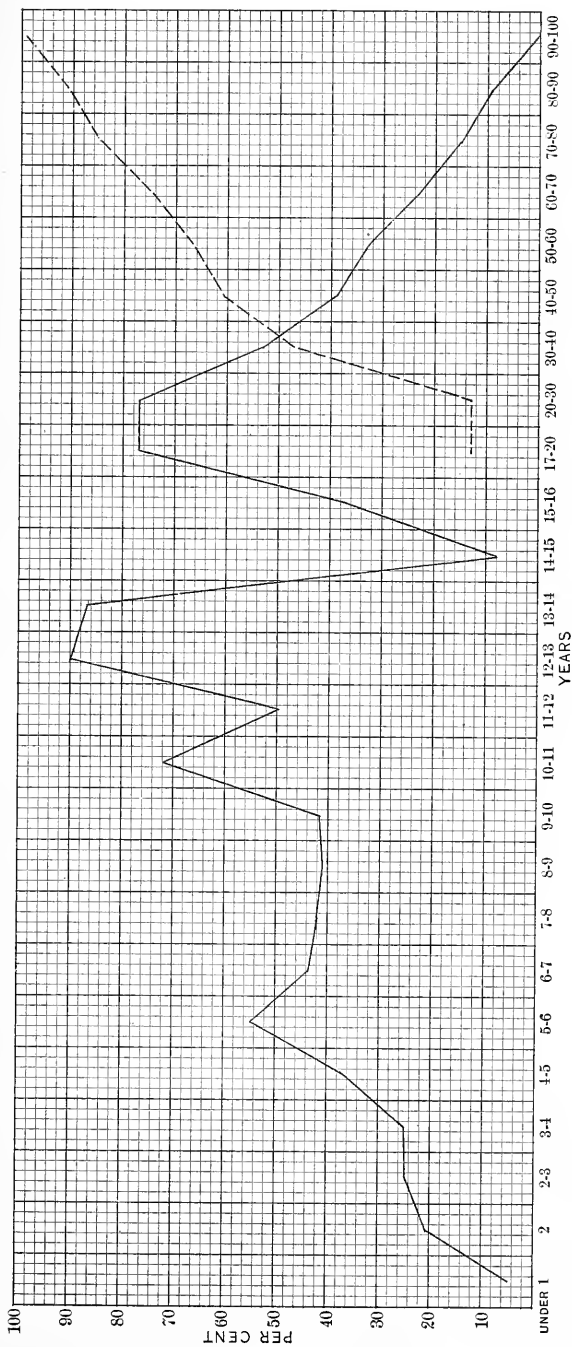


FIG. 1.—Distribution of tuberculous lesions at various age periods as found at autopsies performed by O. Lubarsch. Black line, active lesions; dotted line, latent, quiescent, and healed lesions.

tuberculosis. But Naegeli, Burkhardt and others have pointed out that pleuritic scars and adhesions with underlying pulmonary indurations were considered remnants of tuberculosis only when there were, in addition, found calcified or caseated foci in the lungs or lymph glands, or when tubercle bacilli were found microscopically. It has also been stated by Cornet that these latent lesions were caused by avirulent or mildly virulent tubercle bacilli, perhaps even by non-pathogenic acid-fast microorganisms. But this has not been proved.

The objection has also been raised that the autopsy material obtained in morgues in large cities represents the lowest grades of society, the poorest strata of population, who are most likely to succumb to tuberculosis, while the well-to-do or self-supporting elements of society, even in cities, are by no means tuberculous to such an appalling extent. But it is the poor who present the problem of tuberculosis most acutely. Naegeli also pointed out that his material was not exclusively of the lowest strata of society. Forty per cent. at least were country folk, and 6.5 per cent. were private patients. Moreover, only in 22.5 per cent. was tuberculosis the cause of death, as against 28 per cent. occurring among the general population of the Canton of Zurich, thus showing that the persons on whom he made his autopsies were not excessively tuberculized. In fact, large series of autopsies made in many other cities confirmed Naegeli's findings.

Better confirmation of these findings was, however, supplied by several series of autopsies made on persons who have enjoyed good health but succumbed to accidents or acute diseases. Among 826 autopsies made on such individuals, Birch-Hirschfeld<sup>1</sup> found 171, or 20.7 per cent., with tuberculous lesions. Of these 105, or 12.7 per cent., were healed lesions; 31, or 3.8 per cent., were actively advanced; 35, or 4.2 per cent., were latent or mildly active. Similar results were recently reported by J. G. Mönckenberg<sup>2</sup> who made autopsies on 85 soldiers, fallen in the European War. In 25, or 31.76 per cent., he found distinct evidences of active, latent, or healed tuberculosis. In 5 cases the lesions were so active that they may have been the cause of death, but in the remaining 22 cases the tuberculous lesions were incidental findings.

**Extent of Tuberculous Infection among the Living.**—The extent of tuberculous infection among the living population has been ascertained by the application of the tuberculin test which is even more delicate than the macroscopic examination of the body after death, showing, as it does, the number of persons infected with tubercle bacilli and who have survived or have not at all suffered as a result of the infection. No matter how slight the lesion produced by the tubercle bacilli, the tuberculin test reveals it.

Extensive investigations have been made along these lines, and it was found that there are very few adults living in cities who do not

<sup>1</sup> Deutsches Archiv f. klin. Medizin, 1899, lxiv, 58.

<sup>2</sup> Zeitschr. f. Tuberculose, 1915, xxiv, 33.



react to tuberculin. Those who live in tubercle-laden surroundings hardly ever escape infection. Pollak<sup>1</sup> found that in Vienna 96 per cent. of children of tuberculous parentage were infected before they reached the fourth year of life; Mantoux<sup>2</sup> found that 84 per cent. were infected before they reached the fifteenth year; in New York City the author<sup>3</sup> has found that children living with their tuberculous parents are infected to the extent of 84 per cent. at the age of fourteen, as can be seen from the table and the attached diagram (Fig. 2). Similar results have been obtained while testing large numbers of children of tuberculous parentage in various European cities.

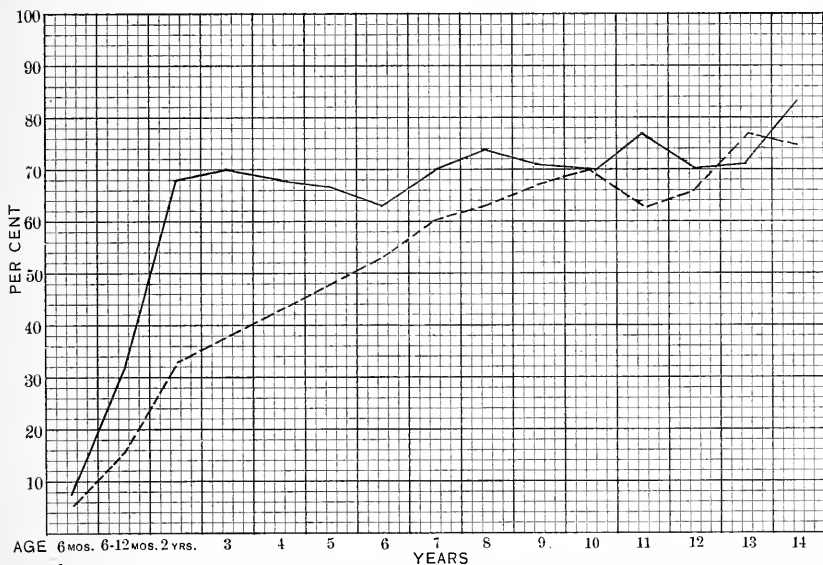


FIG. 2.—Proportion of children reacting to the cutaneous tuberculin test. Black line represents 692 children of tuberculous parentage in New York City; dotted line represents 588 children of non-tuberculous parentage in New York City.

TABLE SHOWING EXTENT OF TUBERCULOUS INFECTION AMONG THE POORER CLASSES IN NEW YORK CITY BASED ON THE APPLICATION OF THE TUBERCULIN TEST ON 1280 CHILDREN UNDER FIFTEEN YEARS OF AGE.

Age.	Children of tuberculous parents.		Children of non-tuberculous parents.	
	Number of cases.	Per cent.	Number of cases.	Per cent.
Under 1 year . . . . .	33	15.15	56	10.07
1 to 2 years . . . . .	49	55.10	39	33.33
3 to 4 " . . . . .	90	68.88	80	41.25
5 to 6 " . . . . .	95	65.26	106	50.00
7 to 10 " . . . . .	244	71.31	173	64.74
11 to 14 " . . . . .	181	74.58	134	69.40
14 " . . . . .	37	83.79	20	75.00

<sup>1</sup> Brauer's Beiträge, 1911, xix, 469.  
<sup>2</sup> Semaine Médicale, 1909, xxix, 371; Presse Méd., 1910, xviii, 10.  
<sup>3</sup> Archives of Pediatrics, 1914, xxxi, 96, 197.

Taking apparently healthy children at random, *i. e.*, those who do not live in homes harboring evidently tuberculous persons, it appears that they are also infected in large numbers. Hamburger<sup>1</sup> found that at the age of fourteen 94 per cent. of the children of artisans in Vienna show signs of infection with tuberculosis. Calmette<sup>2</sup> at Lille, France, testing 1226 persons of all ages taken at random from diverse social strata, all apparently healthy, found that during the first year of life only 9 per cent. were infected, but the percentage kept on increasing, so that at the age of fifteen and over, 87 per cent. were infected. In New York City the author<sup>3</sup> found while testing children of poor, but non-tuberculous parentage, that under one year of age 10 per cent. were infected; between one and two years of age, 33.33 per cent., and the proportion giving positive reactions to tuberculin kept on growing steadily with advancing age so that at the age of fourteen 75 per cent. of "reactors" were found.

It is well known that the von Pirquet test, which was used in these cases, is occasionally negative when applied the first time, but is positive when applied a second or third time. For this reason some who have applied the test but once found a lesser number of reactors. J. B. Manning and H. J. Knott,<sup>4</sup> in Seattle, tested 228 children, aged ten to fourteen years, coming to the Children's Tuberculosis Clinic, the large majority of which were from tuberculous homes. Of 166 with a definite history of exposure 84, or 50.6 per cent., gave a positive von Pirquet test, though 82.1 per cent. of these children showed no clinical evidences of tuberculosis. Of 62 children with no history of exposure 14, or 22.8 per cent., were reactors. But they used only one-half strength of tuberculin, and when found negative after the first application, the test was not repeated. Had they applied it twice or three times, and in full strength, the proportion of reactors would undoubtedly have been higher. George H. Cattermole<sup>5</sup> tested children in Boulder, Colorado, where there is no overcrowding, but plenty of good food and sunshine. Probably one-half the families in Colorado contain one or more adult consumptives. It would be expected that the number of reactors should be quite large. Yet only 38 per cent. were found to have been infected. This anomaly may be explained by the superior social and economic conditions, but it seems to me that the following reasons are more plausible: The number of children was rather small, only 66; if he had extended his investigations the results might have been different; he applied the test but once in most cases, using the von Pirquet and the Moro tests. At any rate it appears that opportunities for infection were not altogether counter-balanced by superior climatic and economic conditions.

<sup>1</sup> Die Tuberkulose im Kindesalter, Berlin, 1913.

<sup>2</sup> Grysez et Letulle, Presse Médicale, 1911, xix, 651.

<sup>3</sup> Archives of Pediatrics, 1915, xxxii, 20.

<sup>4</sup> Amer. Journ. Dis. of Children, 1915, x, 354.

<sup>5</sup> Jour. Amer. Med. Assn., 1915, lxx, 782.

While it is in large industrial cities that tuberculosis is most widespread, as is shown by the high morbidity and mortality from the disease, infection is not lacking in rural communities of civilized countries. Investigations made by Jakob,<sup>1</sup> Hillenberg,<sup>2</sup> Overland,<sup>3</sup> and others have shown that in villages, where a case of open tuberculosis had not been seen for many years, the people living under good economic and hygienic surroundings, and where the milk supply was practically free from tuberculous contamination, 25 per cent. of the school children and about 45 per cent. of the adults gave positive reactions to tuberculin, indicating that they had not escaped tuberculous infection. Here we find that the effect of infection is only an altered reactivity to tuberculin, and not phthisis. The reasons for this phenomenon will be discussed later on.

**Tuberculosis among Primitive Peoples and Races.**—The only regions free from tuberculosis appear to be those inhabited by primitive peoples who have not come in contact with civilization. Thus, the American Indian, before the advent of the white man on this continent, knew nothing of the disease, as was shown by Woods Hutchinson,<sup>4</sup> Hrdlicka,<sup>5</sup> and others. Nor do the savage and barbarian races of Central Africa and Asia seem to have had experience with tuberculosis, until the whites brought it to them. Among these primitive peoples the tuberculin reaction is always negative, and autopsies made on their dead reveal no active or healed tuberculous lesions, as is the case with newborn infants among Europeans. But it appears that as soon as these peoples come into contact with civilized man they are infected in large numbers. This was observed among the American Indians, the native tribes of Australasia and Africa, etc. The application of the tuberculin test among these races by Calmette,<sup>6</sup> Metchnikoff,<sup>7</sup> Zieman,<sup>8</sup> and others has shown clearly that the frequency of tuberculous infection depends directly on their contact with civilization. It is altogether absent or extremely rare among those races who have recently met the white man, but the proportion grows in direct ratio to the intensity of immigration of European settlers, and with commercial interchange between them and civilized humanity. It is also evident that their immunity from this disease before the advent of the white man was not due to racial or climatic conditions, as was suggested by some earlier writers, but solely to the absence of tubercle bacilli, because as soon as these are imported, the natives display a striking vulnerability to the disease, which is greater the longer they have been protected against the importation of tubercle bacilli.

<sup>1</sup> Die Tuberkulose und die hygienische Misstände auf dem Lande, Berlin, 1911.

<sup>2</sup> Tuberkulosis, 1911, x, 254.

<sup>3</sup> Internat. Zentralblatt für Tuberkulose, 1914, viii, 635.

<sup>4</sup> New York Med. Jour., 1907, lxxxvi, 624.

<sup>5</sup> Tuberculosis among Certain Indian Tribes of the United States, Washington, 1909.

<sup>6</sup> Ann. de l'Institut. Pasteur, 1912, xxvi, 497.

<sup>7</sup> Ibid., 1911, xxv, 785.

<sup>8</sup> Centralblatt f. Bakteriologie, 1913, lxx, 118.

**Racial Differences in Susceptibility to Tuberculous Infection.**—

A study of the epidemiology of tuberculosis also teaches that the dangers of tuberculous infection depend on the length of time a people have been exposed to the disease. Thus, when primitive peoples, who have never been affected with this disease come into tubercle-laden surroundings, they are soon infected and the disease runs an acute and fatal course in nearly all cases. This is often the case with savages and barbarians brought to Europe or America: They almost invariably acquire tuberculosis and succumb in a short time. The American Indians, coming in contact with the whites and incidentally with the tubercle bacillus, are being decimated by the disease which runs an acute and fatal course among them, and the same is true of the negro population in this country.

A drastic illustration has been reported by Cummins<sup>1</sup> from Egypt where the Sudanese soldier, recruited from tribes among which tuberculosis is practically unknown, is much more liable to tuberculosis than the Egyptian soldier who has been raised in a region where the disease has been quite common for centuries. In former times slaves of the Sudanese race were the cheapest in the market because it was assumed that a large number would contract the disease and die.

This is exemplified again by the conditions observed among the immigrants to the United States. The Irish and Sicilian immigrants, and to a lesser extent the Hungarians, Slavonians, and Scandinavians, mostly hail from agricultural parts of their native country where they have known very little of tuberculosis. In this country, working in closed factories, and coming in contact with tuberculous fellow-workmen, many soon contract the disease which runs an acute course, terminating fatally in a large proportion of cases. Among immigrants coming from countries or cities where they have been exposed to infection for generations, as is the case with the English, Germans, and especially the Jews, the rates of tuberculous mortality are much lower.

When speaking of race influence on the incidence and mortality from tuberculosis, the facts just mentioned must always be borne in mind. Tuberculosis appears not to be a racial problem—there are no races which are more or less vulnerable to the disease because of their ethnic peculiarities, such as height of the body, color of the skin, eyes and hair, or other somatic or morphological traits which distinguish one race from another. Every human race or ethnic group when first meeting with tubercle bacilli is as vulnerable as another. It is only after they have been exposed for many generations to the disease that they acquire a certain power of resistance against infection which, though occurring in almost everyone who has been exposed to infection, is less liable to cause disease than in races which present virgin soil

<sup>1</sup> Trans. Soc. Trop. Med. and Hyg., 1911–1912, v, 245.

to the bacilli. The mechanics of this acquired immunity will be discussed later on.

## MORTALITY FROM PULMONARY TUBERCULOSIS PER 100,000 POPULATION.

Country.	1861 to 1865.	1866 to 1870.	1871 to 1875.	1876 to 1880.	1881 to 1885.	1886 to 1890.	1891 to 1895.	1896 to 1900.	1901 to 1905.	1906 to 1910.
United States . . . . .									171	147
England and Wales . . . . .	253	245	222	204	183	164	146	132	122	111
Scotland . . . . .	252	262	248	229	211	189	174	165	145	
Ireland . . . . .		183	191	200	208	212	214	213	215	191
Australia . . . . .					122	121	107	94	89	75
New Zealand . . . . .					91	84	81	78	70	62
*Ontario Province . . . . .					125	116	114	141	129	113
Germany . . . . .				361	348	314	224	194	186	159†
Prussia . . . . .				317	312	290	247	208	191	162
Bavaria . . . . .							287	262	243	214†
Saxony . . . . .			251	251	244	236	212	194	154	135†
Baden . . . . .					312	297	278	244	217	183
*Austria . . . . .					377	393	383	394	345	305
Switzerland . . . . .				200	209	213	199	190	189	176†
Netherlands . . . . .							189	165	133	125
*Belgium . . . . .	305	305	335	323	301		165	142	118	102†
France . . . . .							255	249	265	277†
Italy . . . . .						137	100	106	116	123†
Spain . . . . .									148	135
Denmark . . . . .				262	249	231	200	160	149	134†
Norway . . . . .			108	126	140	144	173	206	196	200†
Finland . . . . .		374	414	367	255	256	261	273	291	
Serbia . . . . .							251	231	280	297†
*Hungary . . . . .								364	397	374
Chile . . . . .						235	269			
Japan . . . . .						101	136	145	146	154†

NOTES.—All figures refer to pulmonary tuberculosis, except those marked \* which include all forms of tuberculosis.

Figures in the last column marked † are only for 1906-1908.

**Geographical Distribution.**—Fifty years ago Hirsch in his classical study of *Geographical and Historical Medicine* arrived at the conclusion that tuberculosis is a disease of all times and all countries. With our present knowledge we have not discovered any proofs to the contrary. Observations in every part of the habitable globe show that the presence or absence of the disease is determined less by geographical location or climatic phenomena than by social and economic conditions and, above all, by the presence or absence of the tubercle bacillus. We have shown in the preceding pages that its absence in certain countries has not been due to either an immunity of the population, nor to the climate in which they live, nor to the altitude on which they have been located. Indeed, it is obvious that as soon as the tubercle bacilli are introduced among any people in any geographical location, the disease is not slow in making its appearance. The comparative absence of tuberculosis in the Rockies, the Andes, and other mountainous regions in former times was apparently due to the scarcity of population and the peculiarity of the occupations there pursued. In the mountainous regions of the United States tuberculosis was scarce before consumptives began to immigrate in search of health. Brown, investigating conditions in El Paso, Texas, found that the testimony of physicians is to the effect that deaths due to this disease are rare among the indigenous population; E. A. Sweet<sup>1</sup> finds this to be true of the entire southwest region of this

<sup>1</sup> Public Health Reports, 1915, xxx, 1059, 1147, 1225.

country, and Cattermole confirmed it in Colorado. But it appears that the infection of people living under good sanitary, and above all, economic conditions does not always produce phthisis, especially in regions where outdoor life is the vogue.

DEATH-RATES FROM PULMONARY TUBERCULOSIS PER 100,000 POPULATION  
IN VARIOUS CITIES.

City.	1881 to 1885.	1886 to 1890.	1891 to 1895.	1896 to 1900.	1901 to 1905.	1906 to 1910.
New York . . . . .	398	350	286	242	215	197
Chicago . . . . .	180	177	176	154	152	162
Boston . . . . .	411	377	289	240	217	175
Philadelphia . . . . .	311	269	233	210	215	206
London . . . . .	222	197	185	175	157	132
Edinburgh . . . . .	212	191	180	187	157	114
Glasgow . . . . .	311	250	227	195	170	140
Dublin . . . . .	346	341	335	317	309	268
Belfast . . . . .	382	402	382	329	307	235
Paris . . . . .	441	440	409	379	390	374
Berlin . . . . .	..	..	..	..	..	188
Hamburg . . . . .	..	..	238	200	169	137
Munich . . . . .	389	348	312	303	269	226
Dresden . . . . .	376	334	283	247	224	180
Breslau . . . . .	331	313	342	321	318	271
Amsterdam . . . . .	238	234	204	185	144	138
Rotterdam . . . . .	219	192	188	170	133	127
The Hague . . . . .	199	179	163	160	128	124
Vienna . . . . .	685	576	474	381	336	274
Prague . . . . .	728	609	512	472	525	385*
Budapest . . . . .	715	591	434	376	367	340
Trieste . . . . .	522	491	439	402	396	369
Christiania . . . . .	320	287	282	274	229	183*
Stockholm . . . . .	344	303	269	246	227	230
Copenhagen . . . . .	273	246	198	180	144	136
Petrograd . . . . .	547	449	384	321	305	301
Moscow . . . . .	411	393	391	324	268	258
Milan . . . . .	335	307	284	204	232	220
Turin . . . . .	240	222	250	234	225	183
Sydney . . . . .	193	157	119	98	98	72
Melbourne . . . . .	233	213	182	153	139	109
Montreal . . . . .	282	256	235	250	197	163*
Toronto . . . . .	203	207	242	234	174	
Rio de Janeiro . . . . .	548	..	446	474	455	402

Figures marked \* indicate that the death-rate in the last column is only for 1910.

**Incidence among Rural and Urban Residents.**—Of greater influence than climate and altitude appears to be life in the city as compared with life in the country as regards the morbidity and mortality from tuberculosis. It appears that country dwellers, while not exempt from infection with tubercle bacilli, are yet less likely to suffer from phthisis than the city residents. Thus, the average death-rate from tuberculosis of the lungs in the registration area of the United States during the decade ending with 1909 was 154.7 per 100,000 population, but in the cities of the registration area the rate was 177.4 against a rural death-rate of but 124.1. These differences would be even greater if we excluded the rural centres in which factories, mills, coal mines,

etc., are located and where the workers live to all intents and purposes under the same conditions as those in the cities. These differences in the mortality from phthisis are found in every country where vital statistics are gathered. In England and Wales the mortality per million population was in 1913: London, 1335; England and Wales, 1004; rural districts, 742; all urban districts, 1075. The table on page 64 shows the high mortality-rates from this disease in large cities in various parts of the world. When compared with the rates for the entire country, as given on page 63, the differences are clear.

The establishment of sanatoriums for consumptives in rural districts during recent years has apparently increased the mortality from this disease in certain country districts. Thus, in 1910 the death-rates from pulmonary tuberculosis in the State of New York were: in cities, 165.7; and in the rural districts, 120.1, while in Colorado, the Mecca of American consumptives, the rates were: cities, 288.2; in rural districts, 155.9. It is thus evident that with superior climate and altitude, Colorado has a higher mortality from pulmonary tuberculosis than the State of New York. Of course, the reason is that most of the fatal phthisis in Colorado is imported.

Wherever available, statistics show clearly that there is more fatal tuberculosis in cities than in the country. The reasons for this disparity are to be sought not only in the outdoor life which country dwellers indulge in more than city people, but more in the difference in social and economic conditions.

**Social and Economic Factors.**—There is no question but that infection with tubercle bacilli is to a large extent influenced by social and economic conditions; but it appears from available evidence that the development of phthisis is almost altogether dependent on these factors. Thus, we find among the so-called well-to-do, the cutaneous tuberculin reaction only rarely reveals hypersensitiveness among infants and children. Schlossmann even says that a positive skin reaction is hardly ever found among the children of his rich clientele, indicating that they are free from infection. The experience of American physicians appears to be to the same effect, though we do not have data about inoculation of a large series of well-to-do children in this or any other country. It is, however, a rule among pediatricists to place great reliance on the tuberculin test in children. That this is justified in the case of children of prosperous parentage may be true, but whether in older children a positive skin reaction is exceptional is open to question. When children attend school, and later when they go out into the world, meeting all sorts and conditions of men, they are no longer sheltered against infection, and most of them, in fact, do become infected sooner or later.

The high proportion of positive reactions obtained among children and adults in rural districts in Germany and Scandinavia, where infection has taken place despite the absence of known open cases of tuberculosis, and even where bovine infection could be excluded, appears

to confirm this view. In fact, it is very rare to find an adult in a large city who does not show a positive skin reaction to tuberculin, irrespective of his social or economic condition.

Among the millions of proletariat in large modern industrial cities infection appears to be most rampant. All reliable tests—autopsies and tuberculin—have shown that very few escape infection, and the clinics, sanatoriums and hospitals for tuberculous patients derive their clinical material mainly from these strata of population. A study of the mortality-rates also shows that these are the people who are most likely to succumb to tuberculosis. One has only to glance over the maps of New York City prepared under the auspices of Herman M. Biggs to be convinced that poverty and tuberculosis go hand-in-hand. The blocks inhabited by the rich show exceedingly few deaths from this disease, while those inhabited by the artisans, the laborers and the poor—the “slums”—are appallingly studded with cases of phthisis.

Illustrations from other cities are not wanting. In Hamburg the death-rates from tuberculosis are in inverse ratio to the amount of income tax paid by the various groups of population. In Paris, Bertillon found that in the very rich district Elysée the mortality from tuberculosis is the least in the city; it is somewhat higher in the rich Opera district; higher in the very well-to-do district Luxembourg; higher yet in the well-to-do Temple district; very high in the poor Reuilly district, and highest in the Twentieth Arrondissement, where the inhabitants are exceedingly poor. In Glasgow, according to Glaister, the mortality is higher among families living in one-room apartments than in those who live comfortably in several rooms. In Edinburgh A. Maxwell Williamson<sup>1</sup> found that the number of cases of tuberculous disease increases in proportion as the house accommodations become limited. “Pulmonary tuberculosis is a disease which in 70 or 80 per cent. of cases occurs in houses of three rooms and under; the number of cases is larger in two-room houses than in three; larger in houses of one room than in two; and the number of cases of the disease increases almost in direct proportion to the number of small houses in any district or ward of a city.” The relation of phthisis to overcrowding is seen clearly in the industrial cities of the United States.

Similar investigations as to the relations of wages to morbidity and mortality of tuberculosis have shown that higher wages mean less of the disease (see p. 70). The experience of life insurance companies is to the effect that industrial policy holders who pay small weekly premiums are more likely to succumb to the disease than those who hold “ordinary” policies paying annual premiums. From figures supplied by Dr. Lee K. Frankel it is clear that the experience of the Metropolitan Life Insurance Company is in agreement with the view just expressed. Those who insure their lives for small sums, paying small weekly premiums, technically called “industrial insurance,”

<sup>1</sup> Brit. Jour. of Tuber., 1915, ix, 111.



are more likely to succumb to pulmonary tuberculosis than those who insure in the "ordinary department," for sums not less than \$1000, and paying large annual premiums. During 1914 the death-rates per 100,000 were as follows:

Age period.	"Ordinary" policy holders.	"Industrial" policy holders.
20 to 24 . . . . .	110.32	339.7
25 to 34 . . . . .	99.18	390.4
35 to 44 . . . . .	104.73	352.3
45 to 54 . . . . .	108.95	496.9
55 to 64 . . . . .	106.68	373.5
65 to 74 . . . . .	113.71	219.2
75 and over . . . . .	..	168.2

In Europe it was found that the larger the amount for which the person is insured, the less likely is he to succumb to tuberculosis.

The slums of large cities contain "lung" blocks which have been pictured in such sombre colors in the popular tuberculosis literature. Of course, the bad housing conditions are responsible to a large extent. But it must be remembered also that "a slum is not constituted solely of broken-down houses, but also of broken-down occupants, and it is perhaps easier to remedy the one than the other," says John Glaister.<sup>1</sup>

Thus, we have a vicious circle in the economics of tuberculosis. Poverty brings about congestion and overcrowding, enhancing the chances of massive infection; it also compels its victims to work in unsanitary factories, mills and workshops and at trades which are dangerous in this regard. The vitality is depressed and the powers of resistance reduced as a result of insufficient and improperly prepared food, so that infection more often terminates in phthisis than among those who are higher in the social scale.

However, that the well-to-do and rich do not escape is evident when we glance into the modern private sanatoriums which derive their clientele from those who can pay more than fifty dollars per week, not including medical attendance. The resorts in Europe are also filled with rich consumptives, as can be seen in Switzerland and the Riviera. Of course, this shows that not all well-to-do individuals live wisely, even though they can well afford to do so.

**Influence of Age.**—In considering the influence of age on the incidence of tuberculosis we must again differentiate tuberculous *infection* from *morbidity* and from *mortality*, and also the various forms of the disease.

The newborn infant is free from tuberculosis as we have shown; infection takes place during the lifetime of the individual who is exposed to the bacilli. We have already seen that those living in a tuberculous milieu do not escape, and during the first year about 15 per cent. are infected; during the first five years about 50 per cent., and at the age of fourteen over 80 per cent. are infected. Even children of non-tuberculous parentage are infected with tuberculosis to the same

<sup>1</sup> Practitioner, 1913, xc, 344.

extent as those of tuberculous stock, but not at such an early age, and when reaching adolescence the difference is not so pronounced as would be expected *a priori*.

MORTALITY FROM TUBERCULOSIS IN THE REGISTRATION AREA OF THE UNITED STATES PER 10,000 LIVING AT THE GIVEN AGE AND SEX, 1910-1913.

Age.	Pulmonary tuberculosis.		All other forms of tuberculosis.	
	Males.	Females.	Males.	Females.
0 to 1 . . . . .	6.73	5.68	13.76	12.14
1 . . . . .	4.72	4.00	11.78	10.64
2 . . . . .	2.14	1.97	6.13	5.53
3 . . . . .	1.44	1.41	3.95	3.84
4 . . . . .	1.00	1.16	2.90	2.78
5 . . . . .	0.97	0.94	2.10	1.54
6 . . . . .	0.92	0.84	2.01	1.37
7 . . . . .	0.85	1.19	1.83	1.95
8 . . . . .	0.63	1.26	1.36	2.07
9 . . . . .	0.98	1.31	2.11	2.14
10 to 14 . . . . .	1.22	2.94	1.15	1.35
15 to 19 . . . . .	7.96	11.09	1.72	2.09
20 to 24 . . . . .	16.27	17.66	2.10	2.26
25 to 29 . . . . .	18.98	19.33	2.12	2.10
30 to 34 . . . . .	21.70	18.62	2.08	2.01
35 to 39 . . . . .	23.13	16.22	2.09	1.89
40 to 44 . . . . .	23.47	14.25	2.07	1.69
45 to 49 . . . . .	23.32	11.99	2.02	1.63
50 to 54 . . . . .	21.68	11.19	2.04	1.63
55 to 59 . . . . .	22.99	11.80	2.47	1.96
60 to 64 . . . . .	22.13	12.39	2.56	1.92
65 to 69 . . . . .	21.00	14.25	2.45	2.22
70 to 74 . . . . .	20.11	15.87	2.68	2.37
75 to 79 . . . . .	18.02	16.07	2.41	2.70
80 to 84 . . . . .	13.64	13.24	2.02	2.20
85 to 89 . . . . .	12.48	10.23	2.38	2.23
90 to 94 . . . . .	9.71	6.58	1.21	1.25
95 and over . . . . .	10.37	6.71	..	1.52

The morbidity from the disease is greatly influenced by age. During the first two years of life tuberculosis is very frequently encountered in the form of acute miliary tuberculosis, and tuberculosis of the joints, bones, and glands. Between two and ten years of age we mostly find the milder forms of osseous, glandular, and articular tuberculosis and chronic pulmonary tuberculosis is very rare. Only after the age of ten does the latter form of tuberculosis make its appearance, and after fifteen years of age it becomes the menace of society—the proverbial “white plague”—causing more misery than any other disease.

The disease is, however, for lack of reliable morbidity statistics, best gauged by a study of the mortality-rates. From the above table and page 69 it is seen that there are two maximums of mortality. The first during the first two years of life; while beginning with the third year tuberculosis becomes a very infrequent cause of death until the tenth year is reached, when it again begins to rise, reaching its full height at twenty years, and keeps at that high level with slight fluctuations until sixty years, when there is again a slight decline,

As has been pointed out by Ranke,<sup>1</sup> the rate of infection with tuberculosis does not follow closely the rate at which the disease kills. We have shown above that infection begins during the first year of life, keeps on increasing during every subsequent year until at the age of twenty very few individuals are found who have escaped it. The mortality is comparatively high during the first year of life, but then declines, so that between three and twelve years, just the period when most infections occur, the number of deaths is the least, and only after the fifteenth year does the mortality rise to its highest point and keeps at it throughout life. The bearings of these facts on the problems of phthisiogenesis and prophylaxis will appear in other sections of this book.

**Influence of Sex.**—From the table on page 68 we find that during the first six years of life the mortality from pulmonary tuberculosis is somewhat, though not very materially, less among females than among males. After the sixth year the rates among females are higher than among males of the corresponding age groups. Between fifteen and thirty years of age the difference in favor of the males is striking. After thirty years the females again show lower mortality-rates which keep up until the end of natural human life. The total mortality is less among females than among males, a fact which has been observed in all countries where vital statistics are available. In England and Wales the mortality from phthisis in 1913 was: Among the total population 9.81 per 10,000; among males 11.54, and among females only 8.18.

Various explanations have been offered for this disparity in the mortality from phthisis between the two sexes. It has been suggested that the more hazardous occupations in which men are mainly engaged reduced their resistance and predisposed them to phthisis; or when becoming sick with the disease the chances, of recovering are less in the case of men who have to work for their support as well as for those depending on them. But during the ages of fifteen to forty-five, when menstruation, pregnancies and lactation undermine the resisting powers of women, it would be but natural that the mortality from phthisis should be high among them. Vital statistics in some countries seem to support this view, but in the United States the higher mortality among the women keeps up only until the age of thirty, when it again declines as compared with the men.

It appears to me that the higher mortality from phthisis among women between fifteen and thirty in the United States is to be attributed to the large number engaged in gainful occupations. This is confirmed by the census returns showing that among all classes of population, male and female, ten years of age and over without regard to occupation, the proportion of deaths from tuberculosis is 56 per cent. males and 44 per cent. females. When women enter the occupations

<sup>1</sup> Münch. med. Wchnschr., 1914, lxi, 2099.

to earn a living, as B. S. Warren<sup>1</sup> has shown, the proportion is reversed and the difference much greater. Thus, among salesmen tuberculosis constitutes 15.8 per cent. of all deaths, as against 31.1 per cent. among saleswomen; among silk-mill weavers, men 19.7 per cent. and women 38.3 per cent.; among woollen-mill operatives, males 22.2 per cent. and females 29.2 per cent.; clerks and copyists, males 29.2 per cent. and females 31.8 per cent.; and boot and shoemakers, males 13.3 per cent. and females 31.8 per cent. It thus appears that it is more a problem of industrial conditions than of sexual differences. In fact, women do not bear hard work under deleterious conditions as well as men, and succumb to phthisis in greater numbers when, in addition to their physiological functions, they become bread-winners.

**Mortality-rates from Pulmonary Tuberculosis.**—It is impossible at present to give with certainty the extent of tuberculous morbidity in any population. Even in cities where registration of this disease is compulsory, the data collected in this manner are not complete, and we do not know the exact number of persons suffering from active tuberculosis. The statistics published by certain benevolent and industrial societies are also inconclusive because they concern only certain groups of people, and the results cannot be applied to the general population. Attempts have been made to ascertain the morbidity-rates from tuberculosis by multiplying the number of deaths occurring in a given region by the average duration of the disease. Thus, there annually occur about 160,000 deaths due to tuberculosis in the United States; in Germany over 100,000; in France 70,000; in England and Wales over 50,000, etc. But attempts at multiplying these numbers by the number representing the average duration of the disease and thus finding the actual number of sick have met with failure because there is no agreement as to the average length of phthisis. Indeed, it has been estimated at from one to ten years by different authors.

The extent of the disease is therefore best gauged by the number of deaths it causes in a given population. The table on page 63 gives the mortality per 100,000 population in different countries. When in connection with these figures we bear in mind that one-third of all the deaths during the prime of life, between fifteen and forty, are due to tuberculosis, of which over 90 per cent. is phthisis, we realize the enormity of the problem presented by tuberculosis and the reason why it has been considered the most important of diseases with which humanity has to cope.

The differences in the mortality-rates for the different countries are due to various causes, mainly the intensity of concentration of population in cities, the character of the occupations pursued by the people and other causes which have already been discussed.

**Decline in the Mortality from Tuberculosis.**—Another point brought out by the figures in this table is that the mortality from tuberculosis

<sup>1</sup> Trans. Nat. Assn. Study and Prev. Tuber., 1913, ix, 153.

has been declining in nearly all countries where statistics are available, excepting in Norway, Ireland, Serbia, Spain, France, Italy, Japan, Hungary, etc. This decline is of great significance, and if the exact causes were ascertained we might be in a position to accelerate it so that ultimately the disease could be stamped out altogether.

It will be noted that in England the mortality from phthisis declined since 1861-1865 from 253 to 147 during 1906-1910. A glance at Fig. 3, showing the mortality in 1851 as compared with 1912, proves conclusively that the mortality has declined. The same is true of Scotland, Australia, Germany, Austria, etc. For the United States Frederick L. Hoffman's<sup>1</sup> statistics show clearly that the mortality from tuberculosis in New York, Philadelphia, Boston, etc., has been constantly declining during the past one hundred years.

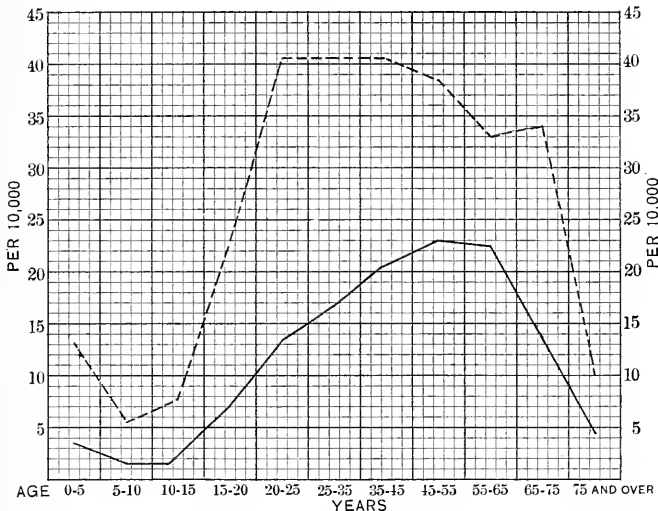


FIG. 3.—Mortality from phthisis by age groups in England and Wales per 10,000 living, showing the decrease from 1851 to 1912. Dotted line, mortality during 1851-1856; black line, mortality in 1912.

What are the causes of this decline in the tuberculosis mortality? All authorities agree that it is mainly due to the causes which have been operative in reducing the general mortality; in banishing or abating the malignancy of most other infectious diseases. Among these factors are largely to be considered the improvements in the sanitary and hygienic conditions under which the bulk of the people live at present. It is also to be considered that modern factory legislation, the improvements in the economic conditions of the people, the shorter hours of work, etc., which are characteristic of the present, as compared with conditions during the first half of the nineteenth

<sup>1</sup> Trans. Nat. Assn. Study of Prev. Tuber., 1913, ix, 101.

century, have been instrumental in reducing the general mortality and of phthisis as well.

**The Effect of the Special Campaign against the Spread of the Disease.**—Most authors when speaking of the reduction in the tuberculosis mortality point at once at the special measures which have been taken to combat this disease as the sole factor in this direction. In fact, the figures compiled in the tables on p. 63 and 64 are always brought forward in proof of the effectiveness of the antituberculosis campaign which has been so aggressively waged.

But careful studies of the available statistical data have not sustained this contention. In England, where the decline has been more pronounced than in any other country, it has been shown by competent statisticians that such is not the fact. Karl Pearson<sup>1</sup> points out that, examining available data, it appears that the death-rates from phthisis are steadily increasing as we go backward to 1838. Now, this could not go on indefinitely because if it did, every individual five hundred years ago must have died in England from phthisis. There was assuredly a time in England when the phthisis rates were rising, just as they have recently been falling. "We have to stretch our ideas of time a little and we should realize the possibility of a typical epidemic curve in the frequency of phthisis. Indeed, the mortality from phthisis in England has been declining since 1838, *i. e.*, long before any special measures had been taken for the control of the disease, or segregation of the sources of infection—tuberculous human beings and animals—had been attempted."

Data from other countries, especially where the disease has become a menace during recent years, confirm these views. During the first half of the nineteenth century there were isolated areas in Europe where tuberculosis was rare, but with the segregation of the population in cities during recent years, and the introduction of modern industrial conditions, the disease has made its appearance and rages there with greater vigor than in countries where the disease has appeared before. Thus, the tuberculosis mortality has been rising in Ireland, Norway, Serbia, Bulgaria, Hungary, Japan, etc., during the same period that it has been declining in England, Germany, etc. There is no doubt that the measures taken for the control of the disease in Norway are as aggressive and advanced as those taken in neighboring Denmark, yet in the former the mortality-rates have been rising, while in the latter they have steadily declined. The same is true of France when compared with Belgium, and similar analogies can be made between other countries, or various regions of any single country.

It appears that the mortality-rates from tuberculosis have been declining to the same extent as the general mortality from all causes, as has been shown clearly by many competent statisticians. Professor Walter F. Wilcox<sup>2</sup> says that "to show that the campaign against

<sup>1</sup> The Fight Against Tuberculosis and the Death-rate from Phthisis, London, 1911, p. 9.

<sup>2</sup> Monthly Bull. New York Board of Health, 1910, xxvi, 85.

tuberculosis is having its effects, it should be found that the death-rates from that disease are decreasing faster than the average for all other causes." But a test of this question with statistics for the mortality in the State of New York shows that the result is a negative one. "No influence of the special campaign can be traced in the figures. The condition in Michigan is similar to that in New York. In Indiana the number of deaths in each instance has decreased, but apparently the proportion of those from tuberculosis to all others has not." In New Jersey and Rhode Island, while the mortality from other causes has been decreasing, that from tuberculosis has been increasing, so that the comparative proportion of the latter has risen. Pearson has proved incontrovertibly that since the campaign has been waged in England against tuberculosis "the rate of fall in the death-rate from phthisis, instead of being accelerated, has been retarded."

Statisticians are not alone in this opinion. In a posthumous paper by Robert Koch<sup>1</sup> he states that the special measures taken for the control of tuberculosis, such as segregation of consumptives, the erection of sanatoriums, etc., are not to be taken as the sole factors which have been instrumental in reducing the mortality from tuberculosis during recent years. He says: "Many have connected the decrease in the tuberculosis mortality with the discovery of the tubercle bacillus. It was stated that after the proofs have been produced that tuberculosis is transmissible, greater care has been taken to prevent infection, while before the discovery of the tubercle bacillus physicians, and with them the laity, denied the transmissibility of the disease. This assumption surely has something in its favor. At any rate, it is a striking fact that, with but few exceptions the decline in the mortality began a few years after the discovery of this bacillus. But just these exceptions prove that the newly engendered fear of the dangers of infection is not the only factor operative in this direction, although we must give it a certain, and not an inconsiderable, amount of credit. Among German authors we often meet with the view that the recent social legislation, especially that concerning workmen's insurance, has been effective in reducing the tuberculosis mortality. To a certain degree there is some correlation in time between these two phenomena in Germany. But, inasmuch as in most other countries such laws have not been inaugurated and the decline in the tuberculosis mortality has taken place to the same extent as in Germany, this factor should also not be taken as a cause."

In this country we now hear similar opinions expressed. William Charles White<sup>2</sup> says: "We cannot possibly avoid the facts that in spite of all our labor our results are not what we might have expected on a right premise; for our reduction in morbidity and mortality from tuberculosis has not kept pace with the reduction in the general death-rate; and, further, our reduction in mortality was about as great

<sup>1</sup> *Zeitschrift für Hygiene*, 1910, lxxvii, 1.

<sup>2</sup> *Trans. Nat. Assn. Study and Prev. of Tuber.*, 1913, ix, 80.

before we started our present methods, and in proving how great the influence of our efforts has been we usually neglect all the influences that operated before we began, and new factors, such as the Mills-Reinecke phenomenon, and ascribe all good to our own work."

**Real Causes of the Decline in the Tuberculosis Mortality.**—Careful study of the economic and social conditions in the various countries where statistical data are available shows clearly that there is a pronounced correlation between urbanization, *i. e.*, concentration of large masses of population in cities, and the death-rates from phthisis. Wherever the process of urbanization is new, wherever modern industries have only recently been introduced, and large numbers of rural population have been attracted to cities, the death-rates from phthisis have been rising. This is the case in Japan, Norway, Ireland, Serbia, Bulgaria, etc., and to a certain extent in Russia, Austria, Italy, France, etc., where the mortality has not decreased perceptibly. On the other hand, in England, where industrial development was operative in the beginning of the nineteenth century, it was at that time that the high phthisis mortality occurred and it began to decline with the adaptation of the people to city life. For this reason the negroes in the cities in the United States, though having a high phthisis mortality, and no special measures are taken to prevent dissemination of the disease among them, also show a strong tendency toward a reduction in the death-rates. Thus, in Baltimore, John W. Fulton found to his amazement that "both races gained against tuberculosis, the whites at the rate of 30.8 per cent., and the negroes at the rate of 24.5 per cent. in the decade of 1904-1913."

We have already shown that whenever people who have hitherto been free from tuberculosis meet with tubercle-laden surroundings, they succumb to the more acute and fatal forms of the disease, while most of those who have for generations been tuberculized are either not harmed by infection at all, phthisis not developing after the vast majority of infections, or when it does develop, it manifests a tendency to pursue an exceedingly chronic course, or heals spontaneously in a large number of cases. The reasons for this phenomenon will be discussed under the heading of Phthisiogenesis (see Chapter V).

The decline in the mortality cannot be attributed to any single cause, but is apparently due to many and complex factors, most of which are obscure at the present state of our knowledge. It seems, however, that recent improvements in the social and economic conditions of the working classes, the inauguration of general hygienic and sanitary measures, and above all the improvement in the housing conditions and in the quantity and quality of the food consumed by the working classes, who are the main candidates for consumption, have all been of assistance in this direction, although the adaptation of the organism to city life, and to the tubercle bacillus, is perhaps of greater importance than all other factors taken together. We must never forget in this connection that the modern methods of prevention aim at but one thing:



the prevention of infection. And in this they have utterly failed, as they should if we consider that hardly 5 per cent. of the open cases of tuberculosis have been isolated. There could not have been more than 90 per cent. of humanity with tuberculous lesions in their bodies as we find at present while making autopsies; there could not have been at any time many more than 75 per cent. of humanity in cities showing conclusive evidence of having been infected with tubercle bacilli when tested with tuberculin. But what has been achieved is a reduction in the morbidity, and especially in the mortality from phthisis even in those who, despite all our efforts at prevention, have been infected with the virus.

## CHAPTER IV.

### FACTORS PREDISPOSING TO THE EVOLUTION OF PHTHISIS.

WE have seen that tuberculosis is a highly transmissible disease; that bacteriological, pathological, and clinical evidence combine to prove that hardly anybody exposed to tubercle bacilli escapes infection. The only difference of opinion among authorities at present appears to be whether as many as 95 per cent. of civilized humanity show evidence that the tubercle bacilli have been implanted in some organs of their bodies, or merely 70 per cent. It is now important to inquire why only 10 or 12 per cent. of humanity succumb to this disease while nearly 90 per cent. remain in good health, in spite of tuberculous infection of which they show undoubted traces. "If, of a large number of persons exposed," says Kingston Fowler, "to infection and infected, only a few acquire the disease, the susceptibility becomes a factor in causation of greater moment than exposure to infection."

Tuberculosis is not a clinical entity like typhoid fever, pneumonia, or smallpox, running a certain course, at times severe, often mild, but always producing the same clinical picture. Tuberculosis in children produces a different clinical picture than in adults. In the former it is usually a bacteremia, affecting the glands, bones, joints, etc., while in the latter it is a local chronic disease of the lungs—95 per cent. of tuberculosis in adults is phthisis pulmonum. How are these phenomena to be explained? Even the evidence which tends to show that milk from tuberculous cattle is responsible for the mild forms of tuberculosis in children, while the human type of bacilli is responsible for the phthisis in adults and the graver forms in children, is insufficient to explain all these remarkable phenomena. The fact that adults consume the same milk is, among others, proof that there are other factors operative in phthisiogenesis.

Another important problem in phthisiogenesis is why do those affected with tuberculosis of the lungs show such different proclivities to suffer as a result of infection with the same type of bacillus? Clinically, we find that some are attacked with the acute forms of the disease, such as acute, general miliary tuberculosis, acute pneumonic phthisis, etc., and succumb in a relatively short time; others suffer from sub-acute phthisis, which may progress slowly or rapidly to a fatal termination, or suddenly take a turn for the better and run a chronic course without any apparent reason to account for the change in the malig-

nancy of the disease; in still others the disease begins insidiously, runs a slow, sluggish course for many years, incapacitating the patient now and then for a variable period, yet he lives indefinitely, perhaps his natural life, and may die from some intercurrent disease. To these must be added the large, in fact the enormous, number of persons in whom the implantation of the tubercle bacilli in the lungs, or any other organ, produces anatomical changes in structure unmistakably recognizable at the necropsy; yet these lesions heal spontaneously, the patient and his physician knowing nothing about the morbid phenomena of tuberculosis during the life of the individual.

What are the factors which endow this last class of persons, who are in the majority among the living, with resisting power that the implantation of tubercle bacilli in their bodies, even causing structural changes in their lungs, does not in the least affect their general health? Which are the factors that predispose others so that when the bacilli are implanted in their bodies the disease runs an acute or subacute course and they sooner or later succumb to the action of these micro-organisms and their toxins?

**Theories of Predisposition.**—Various answers have been given to these questions.

1. Some have seen in the predisposition of patients an expression of heredity; that there are families who are exceedingly predisposed to the action of the tubercle bacilli, while others possess more or less resistance in this regard. In the former infection is followed by phthisis, or tuberculosis of some other organs, which may be mild or severe; while in the latter infection is merely followed by a change in the biological properties of the blood as can be seen from their altered reactivity to tuberculin.

2. Others have attributed the predisposition to phthisis to constitutional, biochemical or serological derangements of the body or the blood. There have even been suggested methods of treatment of the disease along the lines of removing the constitutional defects and thus preventing or curing the disease.

3. Finally, others have maintained that the predisposition to phthisis depends on certain local anatomical peculiarities of the lungs or the thoracic skeleton which reduce the vitality of the organ and thus favor the proliferation of the bacilli which may have been brought there by the air or circulating blood.

We shall discuss these theories in some detail.

## HEREDITY.

**Lack of Reliable Statistics on Heredity of Phthisis.**—The theory of hereditary predisposition may be supported by either statistical data about ancestral tuberculosis, or by biological observations in diseased organisms.

For centuries physicians have noted that in certain families tuberculosis reappears in successive generations, and many patients can trace the disease back to their ancestors and blood relatives. Statistics collected along these lines are plentiful, but on close analysis it appears that they are of little value in proving or disproving the hereditary transmission of the disease, or of a predisposition to it.

Even disregarding the ubiquity of the disease, one out of every seven or eight deaths is due to it, so that it may be found in any large family or its branches, it must be borne in mind that the average history of a tuberculous patient who is derived from uneducated social classes is very unreliable. The statements about the state of health, and especially the causes of death of grandparents, parents, brothers and sisters are open to criticism in the vast majority of cases. Even the questions about their personal history are not accurately answered as a rule. Our patients at the Montefiore Home nearly all state that they had measles during childhood, probably on the principle that everyone must have it. But very few say that they have had diphtheria, typhoid, typhus, scarlet fever, etc., although most of them come from eastern Europe where these diseases are rampant and hardly any attempts are made to check them by proper quarantine regulations, and very few indeed escape. Very few know the cause of death of their parents, hardly any that of their grandparents; in fact it would seem as if their parents were all immune to phthisis, considering that the patients do not mention it after questions are addressed to them on the subject.

In private practice, where we deal with a more intelligent class, we often find that the father has coughed, the mother had hemoptysis, etc., after a categorical answer that there has been no consumption in the family. On the other hand, we know how much suggestion through leading questions suitable for a certain theory may bring out appropriate answers. Many patients are convinced that their blood is not by any means "tainted," that they "come from healthy stock," that "there has never been any consumption in their family," etc.

To prove statistically the hereditary transmission of tuberculosis, or a predisposition to the disease, carefully kept records of many families would be required, in which children of tuberculous parentage have succumbed to the disease despite the fact that they have been removed immediately after birth, thus preventing exposure to infection through intimate contact. This we do not have. Even the data given by orphan asylums, showing that thousands of children of tuberculous parentage do not develop tuberculosis, are of absolutely no value in disproving heredity of this disease. In these institutions children under fourteen are usually kept, and at that age active phthisis is exceedingly rare, as has already been shown.

For these reasons very little confidence can be placed in the statistical compilations of various authors to the effect that among their patients 25, 44.7, or 59.2 per cent. have given a history of tuberculosis

in the parents, grandparents, brothers, sisters, or collaterals. It depends a great deal on the zeal of the questioner to obtain points for the substantiation of his pet theory. Even the excellent statistical studies of Karl Pearson, Weinberg, Schlüter and many others are not at all convincing. In fact M. Burekhardt<sup>1</sup> has found that in non-tuberculous persons tuberculosis in ascendency is just as strongly represented as in the tuberculous, and that the disease in the father is just as frequent in both groups, while the frequent occurrences in the mothers, fathers, brothers, sisters, uncles, and aunts can easily be explained by infection.

**Germinative Transmission.**—The reappearance of tuberculosis in several successive generations is by no means proof that the disease has been transmitted by heredity, nor even that the so-called predisposition to the disease has been inherited. In coal miners the lungs show changes of anthracosis through several generations, so long as they are engaged at that occupation. But no one will say that it has been inherited. Similarly, the social, economic, hygienic, and sanitary conditions and surroundings which were responsible for the phthisis in the parents may be, and usually are, operative in the children who remain in the same social milieu. We may justly speak of social heredity, but not of biological heredity. The latter implied the transmission of characters or their physical foundation, which were contained in the germ plasm, or the parental sex cells. Anything that may affect the fertilized ovum, or affect the embryo, cannot be considered inherited, as was pointed out by Martius,<sup>2</sup> who also shows that intra-uterine infection and germinative transmission of a disease have nothing to do with the problems of heredity, just as extra-uterine influences cannot be considered transmissible.

Experimental investigations by Friedmann show that intra-uterine infection with tubercle bacilli is not impossible. This, in some measure, confirms Baumgarten's theory to the effect that tubercle bacilli may enter the blood stream of the fetus, remain dormant for a long period of years, to flare up again by intense multiplication when for some reason the natural resistance of the body fails. This form of transmission of phthisis cannot strictly be considered germinative heredity—it is actually infection of the fetus from the mother—yet it is important for the clinician, especially to one giving thought to prophylaxis.

Baumgarten<sup>3</sup> bases his theory mainly on experiments with tuberculous chickens. It is well known that the progeny of tuberculous chickens is tuberculous even under conditions when infection after the egg has been laid can be positively excluded. Experimentally it has been found that the albumen of a fertilized egg may be inoculated with tubercle bacilli, and the evolution of the chick goes on as

<sup>1</sup> Zeitschr. f. Tuberkulose, 1904, v, 29.

<sup>2</sup> In Brauer, Schröder, and Blumenfeld's *Handbuch der Tuberkulose*, 1914, i, 395.

<sup>3</sup> *Arb. a. d. Gebiet. d. Path. Anatom. u. Bakteriol.*, 1891–1892, vol. i.

usual; but it develops tuberculosis after it is hatched. This has been done by Baumgarten, Milchner, Gärtner, Maffucci, Koch, and others. The germinative or placental transmission of tuberculosis in which the female ovum, or the male cell, or the complete embryo is infected through the placental circulation with tubercle bacilli, yet keeps on developing, has been proved by other observations, notably in cases in which the newborn infant was found tuberculous. Many such cases have been reported during recent years as occurring in cattle and also in human beings. In fact, localized, calcareous degeneration of some focus in the lungs has been found in newborn infants, showing that they had tuberculosis *in utero* and that the lesions healed.

We are in the dark as to how these bacilli reached the embryo. There are proofs that the female ovum may be infected with tubercle bacilli. Westermeyer, Jani, Jackh, and others have found tubercle bacilli in the human ovary and Spano, Porter, Friedmann, and others have found them in the semen. To be sure, these findings were mostly in persons dead from acute miliary tuberculosis, but it must be borne in mind that individuals with genital tuberculosis often cohabit with the opposite sex and pregnancy is frequent. Indeed, Albrecht, Cavagnis, Maffucci, and others have succeeded in infecting rabbits and guinea-pigs with semen taken from bulls suffering from tuberculosis. Friedmann<sup>1</sup> injected an emulsion of tubercle bacilli into the vagina of rabbits immediately after they had been impregnated by the male. Subsequent observation showed that while the mothers remained free from disease, tubercle bacilli were found in sparing numbers in the seven-day-old fetuses, which were not at all hampered in their evolution. In newborn rabbits whose mothers were thus treated, tubercle bacilli were found in various organs. This tends to prove that spermatogenic infection—*i. e.*, infection with tubercle bacilli brought along with the semen from a tuberculous father—is possible.

But, as has been pointed out by Römer,<sup>2</sup> it can be stated that in general semen contains tubercle bacilli only when the genital organs, especially the testicles, are affected. There is, however, no doubt that genital tuberculosis in the male may be transmitted to the offspring. But it should be emphasized that this must be of exceedingly rare occurrence considering that with each emission millions of spermatozoa are expelled, and that the one on which a bacillus has implanted itself should be just the one that fertilizes the ovum, is a rather remote chance. This mode of infecting the ovum may therefore be left out of consideration.

In fact, Römer,<sup>3</sup> carefully analyzing the results of this and other experimental work along these lines, arrives at the conclusion that so far no unequivocal proof is available in favor of the possibility of infection of the spermatozoa which should remain with sufficient

<sup>1</sup> Zeitschr. f. klin. Med., 1901, lviii, 2.

<sup>2</sup> In Brauer, Schröder, and Blumenfeld's Handbuch d. Tuberkulose, 1914, i, 272.

<sup>3</sup> *Ibid.*, p. 274.

vitality to impregnate the ovum, and a tuberculous, yet living, infant should be born and continue alive for some time.

**Placental Transmission.**—But there is another possibility, namely, intra-uterine infection of the healthy fetus from a phthisical mother during pregnancy; the tubercle bacilli entering by way of the placental circulation. That the placenta may harbor tubercle bacilli is well known; the frequent bacteremia in phthisis explains it. Lehmann, Runge, Nowack, Auché, Chamberland, and many others have found tubercle bacilli in the human placenta. On carefully examining the histology of the placenta of phthisical pregnant women, Schmorl and Geipe<sup>1</sup> found tubercle bacilli in 9 out of 20 cases. In 1 of the 9 the mother had merely an incipient apical lesion. Schmorl estimates that 50 per cent. of pregnant phthisical women have tubercle bacilli in their placentas. He maintains that tubercle bacilli may enter the placenta during any period of pregnancy, and in any stage of the disease, but that they are mostly found in the advanced stages of phthisis and in acute miliary tuberculosis. The fetus may be infected from the mother during the act of birth when vigorous contractions of the uterus may lacerate some of the less resisting parts of the placenta. Infection of the fetus may also occur earlier. That they should enter directly into the fetus is a remote probability, if at all possible, but the bacilli may be brought to the fetus by the blood through the umbilical vein; or by way of the intestine after they have reached the amniotic fluid and were then swallowed or aspirated by the fetus. There have been found tubercle bacilli in the organs of newborn infants which showed no macroscopic or microscopic tuberculous changes.

But very few cases of congenital tuberculosis have been found despite the fact that they have been carefully looked for. Péhu and Chalier<sup>2</sup> found only 51 authentic cases on record in medical literature. It may be added that most of the cases were not conclusively proved. Römer<sup>3</sup> knows of but 30 cases and some of them may be said to be reliable only "in all probabilities." Péhu and Chalier believe that in these cases infection usually takes place at the end of pregnancy when the placental circulation is established and results from a bacteriemia which is usually a terminal event. They should therefore be regarded as examples of transplacental heredocontagion and not of direct heredity.

It is thus shown that, theoretically, placental transmission of tuberculosis is possible. But all available facts combine to prove that it is exceedingly rare among human beings. Indeed, when compared with the large number of infections after birth, the few recorded cases of congenital tuberculosis sink into insignificance. After all, when it does occur at all, it is from mothers who are in the far-advanced stages of phthisis, or who have tuberculous disease of the genito-

<sup>1</sup> Ziegler's Beiträge, ix, 428; Münch. med. Wehnschr., 1904, p. 1676.

<sup>2</sup> Arch. de Méd. des enfants, 1914, xvii, 721.

<sup>3</sup> Loc. cit., p. 276.

urinary system. Such women only rarely conceive, and when they do, abortion is the rule. It is a fact worthy of note in this connection that numerous examinations of stillborn fetuses from phthisical mothers have not revealed any traces of tuberculous infection; even inoculation experiments have failed.

Among cattle congenital tuberculosis appears to be more frequent than among humans. Still, the application of the well-known Bang system has shown that even here it is exceedingly rare. In the United States Harlow Brooks<sup>1</sup> has shown that when calves are removed from their tuberculous mothers immediately after birth, they do not develop the disease.

**Clinical Facts of Heredity.**—Many authors have observed certain clinical phenomena which cannot be explained otherwise than by heredity, either of the disease or of a predisposition to it. Brehmer, and after him several other writers, found that in many cases the onset of the disease occurs at the same age in parents and children. Piéry found that in many families the children mostly succumb before attaining the age of sixteen. While many cases can be cited in substantiation of these observations, it appears that so far a sufficient number has not been collated to prove their significance conclusively.

Of greater moment is the inheritance of the *locus minoris resistentiæ*, which Brehmer described long ago and Turban,<sup>2</sup> Baldwin,<sup>3</sup> Moeller, Kuthy,<sup>4</sup> and others have confirmed. It appears that when pulmonary tuberculosis occurs in parents and children, the chances are immense that the same side of the chest should be affected in each case. This family resemblance in phthisis has been found in about 75 per cent. of cases. In my own experience I also observed that in about two-thirds of cases the side affected was the same in the several affected members of the family. Moeller<sup>5</sup> points out that when a child suffers from a tuberculous lesion of some bone, the chances are that when its brother or sister develops tuberculosis it will also be a disease of bone and not of the soft tissues. These facts are explained by the assumption that some organs or tissues in the body lack powers of resistance, and that this defect is transmitted by heredity. This will be discussed again when speaking of the thoracic anomalies and their relation to phthisiogenesis. Meanwhile it may be stated that these problems have not received the careful study they deserve.

**Disturbances in the Metabolism as Predisposing Factors.**—In the search for the factors predisposing to phthisis many have looked into the metabolism of the body, stating that tuberculous infection is harmless in the vast majority of persons, as long as the metabolic processes are normal; only when certain disturbances occur in this regard can phthisis develop. Some excellent investigations into the

<sup>1</sup> Amer. Jour. Med. Sci., 1914, cxlviii, 718; Trans. Soc. Exper. Med. and Biol., 1914, xi, 50.

<sup>2</sup> Zeitschr. f. Tuberk., 1900, i, 30.

<sup>3</sup> Yale Med. Jour., 1902, p. 215.

<sup>4</sup> Zeitschr. f. Tuberk., 1913, xx, 38.

<sup>5</sup> Lehrbuch d. Lungentuberkulose, Berlin, 1910, p. 30.



functions of the internal secretion of the ductless glands have brought no positive results so far. At any rate, we do not know at present that disturbances in the structure or functions of the thyroid, pituitary, or suprarenal glands have an influence in enhancing the growth of tubercle bacilli in the body. It is, however, a fact that in the enormous literature on the subject of tuberculosis, we cannot find an exhaustive study of the metabolism of persons affected with the disease, and hardly anything about the metabolism in the so-called pretuberculous stage.

Several authors have maintained that an excessive excretion of calcium in the urine can be found in all cases of phthisis long before the onset of the disease. In this country Croftan,<sup>1</sup> Russell, and others have made some studies along these lines, and several French authors—Robin,<sup>2</sup> Binet, etc.—have found that in the pretuberculous stage there is a pronounced excess in the excretion of inorganic salts in the urine, notably those of lime and magnesia. The result is that the blood, bones, and lung tissues show a distinct lack in these mineral salts. Gaube found that the descendants of phthisical subjects excrete on the average more calcium and magnesia than those of healthy stock. Robin sees in this lime and magnesia starvation an excessive amount of self-combustion, and he considers this anomaly in the metabolism the main element in the preparation of the soil prone to tuberculosis, whatever the remote cause may be—heredity, alcoholism, malnutrition, overwork, etc. Infection alone is insufficient to produce phthisis, as is evident from the fact that most people infected with tubercle bacilli escape the disease. It is only when the soil is prepared by the dissimulation and emaciation, by pretuberculous decay, as Robin calls it, that phthisis may develop. The gravity of the pulmonary lesion goes hand-in-hand with the degree of lime starvation, demineralization and emaciation of the body. According to these writers, phthisis is preventable. Demineralization of the body must be sought and, when discovered, prevented by the administration of remedies tending to replace the lime and magnesium which are being eliminated from the body excessively.

These and other findings about the metabolism in phthisis have not been confirmed by all who have made careful studies along these lines. It appears that in the vast majority of consumptives the metabolism is quite normal as long as there is no high fever. The occasional lapses in the metabolism are explained by the usual causes of morbid phenomena observed in other diseases characterized by fever, emaciation, debility, etc. At any rate, this subject has not been studied sufficiently to permit making generalizations.

**Anatomical Peculiarities Predisposing to Phthisis.**—The hereditary and constitutional factors discussed above may explain some of the phenomena of tuberculous disease, but they fail to give an adequate

<sup>1</sup> Sixth Intern. Cong. Tuberc., 1908, i, 275.

<sup>2</sup> Traitement de la tuberculose, Paris, 1912.

explanation for all the cases of phthisis which are met with in practice. For these reasons many authors have suggested that local and anatomical peculiarities are responsible for the liability of the lung apex to tuberculous degeneration.

Various hypotheses have been promulgated with a view of explaining why phthisis is localized in nearly all cases in adults in the apices of the lungs. Some have suggested that the determining factor is the blood content of these organs. It is shown that in congenital heart disease, pulmonary stenosis, which is characterized by oligemia of the lungs, nearly all patients succumb to pulmonary tuberculosis. On the other hand, in diseases of the left heart, especially in mitral stenosis, which are characterized by hyperemia of the lungs, phthisis is very rare. It has also been found that in the upper parts of the lungs the blood and lymph currents are slower than in other parts, and thus embolic deposits of bacilli are favored, no matter by which channel they have entered. Calmette's experimental investigations (see p. 47) seem to confirm this view. Then it must be mentioned that the uppermost three ribs show lesser respiratory excursions than the lower ribs. The result is a slower air current in the upper part of the lung and foreign bodies brought in by the inspired air are retained in the apex. But these and many other hypotheses have failed to adequately explain the apical localization of phthisis, especially now, since we know that infection takes place during childhood, while the evolution of the disease begins after maturity of the patient, as a rule.

**Freund's Theory of Stenosis of the Upper Thoracic Aperture.**—About fifty years ago Freund<sup>1</sup> pointed out that stenosis of the bony thorax is very frequently encountered in consumptives, but his observations were neglected and soon forgotten, to be taken up again by himself, Hart and Harras,<sup>2</sup> and others. Bacmeister's<sup>3</sup> experimental investigations have finally given great plausibility to Freund's theory.

The deformity of the upper thoracic girdle, which may be congenital or acquired, consists mainly in an ossification of the first costal cartilage and a shortening of the first rib which exerts pressure upon the lung apex which it surrounds, thus obstructing the circulation of the blood and lymph and preventing the removal of any foreign body—the tubercle bacilli—that may be brought there by the blood or the inspired air, and favoring its localization at this point. Shortening of the first costal cartilage also involves an excessive inclination of the upper thoracic aperture toward the spinal column. The sternum lies too deeply, the ribs run slantingly downward, the shoulders hang low and forward, the scapulæ protrude like wings, and the result is the phthisical chest of the classical authors.

Freund, Hart, and Harras have studied the tuberculous thorax

<sup>1</sup> Beitr. z. Histologie d. Rippenknörpel, Breslau, 1858.

<sup>2</sup> Der Thorax phthisicus, Stuttgart, 1908.

<sup>3</sup> Die Entstehung der menschlichen Lungenphthise, Berlin, 1914.

on the autopsy table and in the living with the aid of radiography, and have found that stenosis of the upper aperture is very frequent. The abnormal shortening of the first rib makes the transverse diameter short, converting the human thorax into one like that of the lower

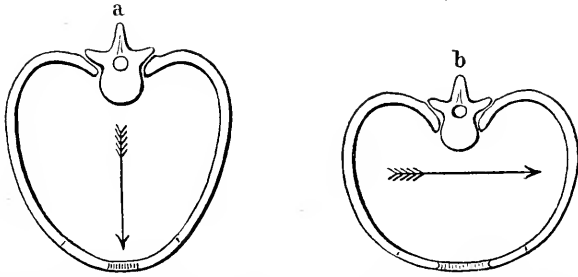


FIG. 4.—Diagrammatic representation of the upper aperture of the thorax: *a*, the primary form (animals, primitive human form); *b*, secondary form (adult man). (After Wiedersheim.)

animals, and to a certain extent infantile, as is shown in Fig. 4. The narrowing usually occurs at the lateroposterior bulging, exactly where the apices of the lung are surrounded by the first rib, which under these conditions compresses the pulmonary tissues beneath. This deformity may occur unilaterally or bilaterally, but the end-

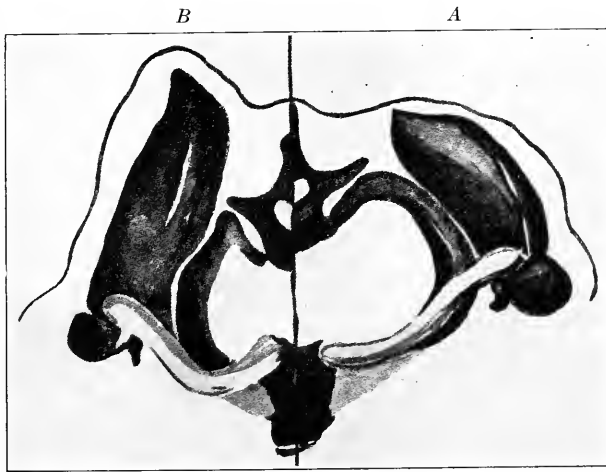


FIG. 5.—Upper aperture of the thorax: *A*, normal on left side; *B*, narrowed at the right. (Freund.)

result is always the same—narrowing and rigidity of the upper thoracic girdle with resulting compression of the lung.

Independent of Freund, Schmorl<sup>1</sup> found a groove about 2 cm. below

<sup>1</sup> Münch. med. Wochenschrift, 1902, xlviii, 1995.

the highest point of the apex of the lung. This groove is very frequently encountered in newborn infants, but in them it can be obliterated when the lung is inflated. During adolescence it disappears in persons with normal chest walls. In most persons in whom it persisted Schmorl found tuberculous lesions beneath the point which was pressed upon by the shortened rib (Figs. 6 and 7).

These observations have been confirmed by Birch-Hirschfeld<sup>1</sup> from another point of view. While searching for the initial lesion of tuberculosis in cadavers dead from other diseases, he found that phthisis



FIG. 6.—Right lung. (Hiss's model.) The indentations made by the ribs are shown. The first groove is the indentation made by the first rib and is known as Schmorl's groove.



FIG. 7.—Left lung. The groove of the first rib is shallower than in the right lung.

begins in the walls of a bronchus of the third to the fifth order, and ascribed it to certain pressure exerted on these tubes, preventing the exit of air and secretions. This bronchiole, which Clifford Allbutt calls "Hirschfeld's bronchiole," from its position and nature, favors that secretions, instead of clearing themselves automatically, will stagnate more or less if pressed upon to a greater or lesser degree by the first rib, located as it is on the apex, leading spirally against the action of gravitation upward, outward, and backward.

<sup>1</sup> Deutsches Arch. f. klin. Medizin, 1899, lxiv, 58.

Finally, Baemeister's<sup>1</sup> investigations have apparently confirmed these anatomical, pathological, and clinical findings. He surrounded young and growing rabbits with a wire loop at the first costal ring, thus causing stenosis of the upper aperture of the bony thorax. The pulmonary apex was thus compressed, and a groove was indented in the lung beneath the wire loop corresponding to the one observed by Schmorl in human consumptives. Infecting these animals, he produced isolated and localized pulmonary tuberculosis, while in normal animals, used as controls, infection produced miliary tuberculosis, but never localized tuberculosis of an apex. In this manner he could produce local tuberculous lesions on either side of the chest, or bilaterally.

There is considerable evidence in support of this theory. In children the upper aperture of the thorax is very elastic, and therefore apical phthisis is exceedingly rare; when infected, the tracheobronchial glands are affected, or general miliary tuberculosis is the result. During the period of puberty, when the spinal column grows and raises the upper thoracic girdle, permitting the first rib to exert pressure on the pulmonary apex, typical phthisis may occur. The largest number of cases of active tuberculosis of the lung, though not the largest number of deaths due to this cause, occur between fifteen and thirty years; between thirty and forty the proportion diminishes, and between forty and sixty there again occur a large number of cases. Hart explains these phenomena in this manner: During puberty and soon thereafter any congenital or acquired shortening of the first rib becomes dangerous to the individual because the growing apex of the lung finds itself hemmed in the small thoracic cavity and the shortened first rib compresses it, thus favoring tuberculous degeneration. After forty, when ossification of the costal cartilage is, to a certain extent, normal, conditions are again favorable for the development of phthisis.

While several authors have confirmed Freund's and Hart's findings, others, like Schulze and Smith, have looked for stenosis in the upper aperture of the thorax while making autopsies on tuberculous subjects, and could not find it in as large a proportion of cases as Freund and Hart reported.

Arthur Keith,<sup>2</sup> Stiller,<sup>3</sup> and other authors are inclined to look upon this deformity of the thoracic girdle rather as a result of tuberculosis than a cause of it. Pottenger<sup>4</sup> points out that the muscle changes described by Freund as hypertrophic and due to overwork, caused by the muscle pulling against an ankylosed rib, is more likely a contraction of the muscle caused by the inflammation within the lung reflexly through the spinal cord. It is also probable, according to Pottenger,

<sup>1</sup> Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1913, xxvi, 630.

<sup>2</sup> Further Advances in Physiology, 1909.

<sup>3</sup> Berl. klin. Wehnschr., 1912, xlix, 97.

<sup>4</sup> Muscle Spasm and Degeneration, St. Louis, 1911.

that the cause of the ossification of the cartilage and ankylosis of the costosternal and sternomanubrial articulations is also a reflex. "The contraction of the muscles covering the apex, together with the limited motion on the part of the diaphragm which is present in even small pulmonary lesions, together with the decreased expansibility and lessened elasticity of the parenchyma of the underlying lung, caused by the inflammatory process within, are causes of lessened motion at the apex; and that these conditions, together with the trophic changes which occur in the bone and cartilage as a result of the reflex stimulation of the nerves which supply these structures, favor ankylosis and ossification."

Of course, the suggestions made by several authors that operative interference is indicated in cases with stenosis of the upper aperture of the thorax for the prevention or cure of phthisis, is rather premature. But it appears that among the many predisposing causes of this disease, the thoracic anomaly just described may play an important role. At any rate, it is worth while to continue investigations along these lines.

**Diseases of the Respiratory Tract as Predisposing Factors.**—Of the diseases which have at one time or another been considered predisposing to phthisis, those affecting the respiratory tract are nearly always mentioned as preparing a favorable soil for the growth of tubercle bacilli. Thus, we occasionally meet with cases of bronchiectasis, syphilis, actinomycosis and cancer of the lungs and chronic pneumonia, in which tuberculosis is implanted at the site of the primary disease. There are two plausible explanations for these phenomena: In most cases it is, in all probabilities, an old, dormant tuberculous lesion, dating back to childhood, that is reawakened into activity by the new disease, assisted by the reduction in vitality and resisting power of the patient. In pneumokoniosis the non-tuberculous lesion in the lung produces a local ischemia, obstructs the lymph channels, and thus prevents absorption or destruction of any tubercle bacilli that may be brought in by the air stream. Pure lobar pneumonia is hardly ever followed by phthisis and, in most cases in which it was said to have been observed, the probabilities are in favor that the primary disease was acute pneumonic phthisis which had subsided and followed the course of chronic phthisis. Especially is this the case with apical pneumonia, and basal phthisis, and many of the so-called "unresolved pneumonias" have been tuberculous from the start.

**Pleurisy.**—Of greater importance is the etiological relation of pleurisy to phthisis. Of course, the secondary pleurisies, those occurring in cases of thoracic neoplasms, cardiac and renal disease have no significance in this regard. But the forms of acute and chronic pleurisy which have been formerly considered "idiopathic," appear to be, in the vast majority of cases, of a tuberculous nature, though many are undoubtedly rheumatic.

This important fact was first ascertained in this country by Vincent

Y. Bowditch,<sup>1</sup> of Boston, who found that out of 90 cases of acute pleurisy which had been observed by his father and followed up by himself between 1849 and 1879, 32 died of, or had, phthisis. Many others, notably Landouzy, Vaillard, Kelsch, Osler, and König<sup>2</sup> have also shown that between 50 and 80 per cent. of patients with pleurisy develop phthisis subsequently.

In the histories of tuberculous patients we often elicit that pleurisy preceded the onset of phthisis. Allard and Köster<sup>3</sup> analyzed 2123 cases of phthisis and found in 650 a history of idiopathic pleurisy. These authors conclude that 47.7 per cent. of cases of idiopathic pleurisy sooner or later develop phthisis; in the cases of dry pleurisy the percentage was 42. On the other hand, von Ruck and Bosanquet found only 5 per cent. of consumptives with a history of pleurisy. Clifford Allbutt,<sup>4</sup> in a review of the subject, arrives at the same conclusion.

It appears that in the young, under fifteen years of age, the proportion of pleurisies that are of a tuberculous character is less than in adults. In fact, only exceptionally does tuberculosis develop in a child after it has passed through an attack of dry or wet pleurisy. Perhaps the same factors are operative which prevent the development of phthisis in all young persons before fourteen years of age, though tubercle bacilli are rare in the exudates in the young.

Strictly speaking, pleurisy cannot be considered as a predisposing cause of phthisis, because it appears that it is essentially tuberculous. Tubercle bacilli have been found in the exudates of between 50 and 80 per cent. of cases, and, since the antiformin method has been used, the proportion is even higher. It is practically established that most cases of "idiopathic" pleurisy are caused by tubercle bacilli. This means that it is not predisposing to phthisis, but that patients with pleurisy are actually tuberculous from the start.

On the other hand, it appears from available clinical evidence that pleurisy has a remarkably favorable influence on the tuberculous process in the lungs. In far-advanced cases of phthisis the disease may be arrested with the onset of pleurisy, especially if an effusion occurs; in many cases we have observed a cure. Some believe that in such cases it was the mechanical influence of the effusion, or the pain restricting the respiratory excursions of the affected side, that afforded rest to the lung and thus favored the healing process, as is the case with an artificial pneumothorax. But this does not explain all cases, and some are inclined to attribute the favorable influence of pleurisy on phthisis to complex biochemical processes.

**Diseases of the Upper Respiratory Passages.**—We often meet with persons who have suffered for years from frequent "colds," showing inflammatory changes in the nose, rhinopharynx and pharynx, recurrent bronchitis and tracheitis, and finally tuberculosis develops. Espe-

<sup>1</sup> Trans. Amer. Climatol. Assn., 1889, vi, 1.

<sup>2</sup> Zeitschr. f. Tuberkulose, 1911, xvii, 521; xviii, 417.

<sup>3</sup> Hygiea, 1911, lxxiii, 1105.

<sup>4</sup> Lancet, 1912, ii, 1485.

cially in children with chronic nasal catarrh or adenoids, tuberculosis has been stated to be very frequent. The fact that these young subjects often have enlarged cervical glands has contributed to the assumption of their predisposition. As a manifestation of the traditional "scrofula" also these morbid phenomena have been considered as in themselves tuberculous.

Microscopic studies of tonsils and adenoids, and inoculation experiments by Walsham, Dieulafoy, and others have shown that tubercle bacilli are often found in these organs. But it appears from all available evidence, clinical experience, animal experimentation and histological studies of tonsils and adenoids, that they by no means contain tubercle bacilli in such large numbers as to be a cause of tuberculosis, and as a point of entry of the bacilli into the body, the pharyngeal tonsil plays only a subordinate role, if any.

**Pulmonary Emphysema and Asthma.**—Of interest is the relation of pulmonary emphysema and asthma to phthisis. Rokitansky said that pulmonary emphysema and tuberculosis occupy a relation of mutual exclusion; and Trousseau considered asthma and tuberculosis as an expression of the same diathesis. Asthmatic patients may bring forth tuberculous children, and conversely, tuberculous parents may have asthmatic children. Brügelmann says that the contrary is true—as long as one has asthma he is immune to tuberculosis, and S. West<sup>1</sup> is of the opinion that "phthisical patients very rarely suffer from spasmodic asthma, and if an asthmatic patient becomes phthisical, an event which is by no means common, the asthma usually disappears." This is in agreement with the view of F. A. Hoffmann,<sup>2</sup> who says that when the two diseases combine, each gives up a part of its peculiarities; the asthma, its characteristic paroxysmal character—the attack becomes weak and indistinct and passes over into indefinite dyspneic conditions; the tuberculosis, its progressive character—it is prolonged and degenerates into fibroid phthisis. The same author considers an emphysematous lung as a distinctly unfavorable soil for the development of tuberculosis.

My own experience leads me to agree only partly with these views. True bronchial asthma is only rarely complicated by phthisis, in fact I have hardly seen half a dozen cases in which this has happened. The paroxysmal attacks of cough and dyspnea seen in some consumptives have often been mistaken for asthma, but a careful consideration of the history and symptomatology of the case shows that they are but pseudo-asthmatic attacks, encountered almost exclusively in fibroid phthisis, and at times in cases of acute pneumonic phthisis. It is different with pulmonary emphysema. I have seen many cases of emphysema complicated by tuberculosis, particularly in workers at dusty trades, garment workers, furriers, rag-pickers, etc. It appears,

<sup>1</sup> Diseases of the Organs of Respiration, London, 1909, p. 600.

<sup>2</sup> In Nothnagel's Practice, Amer. ed., Disease of the Bronchi, Lungs and Pleura, 1903, pp. 241, 291.



however, that the tuberculosis pursues, as a rule, an exceedingly mild course and is very difficult of diagnosis, excepting by a microscopic examination of the sputum.

In this connection it is well to bear in mind the difference in the ages at which these two diseases are most likely to occur: Phthisis is mostly a disease of adolescents and adults before thirty, while emphysema is mainly seen in persons over forty years of age. It is in the latter class that tuberculosis often develops in an emphysematous lung. Emphysema is also frequently seen in chronic phthisis which has healed, and also in the unaffected lung or parts of the lung in patients with active phthisis.

The reasons why asthma and emphysema are some protection against phthisis are not clear. Some are inclined to attribute it to the atrophic condition of the pulmonary parenchyma which renders it unfavorable for the growth of the bacilli; others believe that because the inspiratory current is slow and inadequate, it cannot bring bacilli deeply into the lung. Perhaps the venous hyperemia, which is present in most cases of emphysema, prevents the development of phthisis like certain forms of heart disease do.

**Diseases of the Heart and Bloodvessels.**—Diseases of the heart have also been found etiologically related to the development of phthisis. Louis,<sup>1</sup> in 1836, pointed out that the heart of the consumptive is small, and ever since considerable evidence has accumulated showing that the size, capacity and thickness of the walls of this organ are usually smaller in the consumptive than in healthy persons. Many authors even consider a congenital hypoplasia of the cardiac muscle a prerequisite, or at least a predisposing factor, in phthisis. That an hypertrophied heart is exceedingly rare in phthisis is well known to all who have examined chests with the aid of radioscopy, or made autopsies on persons who died from tuberculosis.

Careful pathological research has, however, shown that in the incipient stage the heart is of normal size and that with the progress of the disease it participates in the wasting process of the organs of the body, especially the muscles. In other words, the small heart is an expression of the general cachexia of phthisis, a phenomenon often observed in other wasting diseases, notably cancer. But even this is denied by some competent observers. Sir Douglas Powell<sup>2</sup> says: "I have always held the belief that the heart in pulmonary tuberculosis did not partake in the wasting of other muscles, and although perhaps not absolutely of normal weight, was yet relatively, or perhaps more than relatively so, in relation to the body weight. My impression clinically, too, is that the right side of the heart is relatively somewhat enlarged and thickened in the chronic forms of the disease."

On the whole it can be stated that a small heart is not a predisposing factor in phthisis, as has been assumed by some authors. Even the

<sup>1</sup> Recherches anatomico-pathologiques sur la phtisie, Paris, 1825.

<sup>2</sup> Lancet, 1912, ii, 1415.

suggestion that a small heart may cause relative anemia of the lungs does not hold, as a rule, because, while it is true that with each beat a lesser amount of blood is propelled to the lungs, this, however, is compensated by the greater frequency of the heart beat.

But a large and hypertrophied heart appears to a certain extent a protection against the development of phthisis. This is seen in the case of valvular disease, especially of the left side of the heart. As far back as 1844 Rokitansky<sup>1</sup> asserted that diseases of the heart and blood-vessels producing passive congestion of the lungs are a preventive of phthisis. Traube later modified this law by saying that only mitral stenosis excludes phthisis, while in aortic disease tuberculosis is occasionally met with. Fagge also held that mitral stenosis is almost a complete bar to tuberculosis, the postmortem records of Guy's Hospital supplying only 4 cases in the course of thirty years. Percy Kidd's<sup>2</sup> statistics give only 1 instance in 500 cases, and Walsham's, 1 in 130 cases.

Inasmuch as this point has lately been contested in this country by Norris, Burns, and others, it is worth while to find out what autopsies made in recent times have revealed. Among 4359 autopsies performed by Birch-Hirschfeld, he found that 907, or 20.8 per cent., presented lesions of chronic pulmonary tuberculosis; among 107 with valvular lesions, only 5, or 4.6 per cent., showed tuberculous lesions in the lungs, and of these the heart defect was in the pulmonary valve in 2. In other words, only 3 cases of mitral disease with tuberculosis were found in this large material. Norris<sup>3</sup> collected from the literature records covering 8154 autopsies on tuberculous subjects where only 3.5 per cent. showed signs of valvular heart disease. While personally performing 1764 autopsies on tuberculous subjects he found 130, or 7.3 per cent., of valvular disease. Anders<sup>4</sup> calculated only 1.2 per cent. in 10,687 autopsies, and Brown<sup>5</sup> collected figures of 71,115 autopsies with but 0.9 per cent. of valvular heart disease in phthisis. Statistics like these show more conclusively than clinical observations the rarity of phthisis with mitral defects. Endocarditis may occur in the course of phthisis, as a complication, but in the majority of cases it appears after the onset of tuberculosis; it only rarely precedes it. As a terminal affection it is not rare, and then is usually due to staphylococci, streptococci and is, as a rule, verrucose in type. Tuberculous endocarditis does occur, but it is exceedingly rare.

Murmurs in phthisical subjects do not mean endocarditis, as a rule. They are usually due to fatty degeneration of the heart with dilatation, pleuropericardial adhesions, cardiac displacement, etc. The latter may even produce arrhythmia. C. M. Montgomery<sup>6</sup> found murmurs

<sup>1</sup> Handbuch der patholog. Anatomie, Vienna, 1844, ii, 520.

<sup>2</sup> St. Bartholomew's Hosp. Rep., xxiii, 239.

<sup>3</sup> Amer. Jour. Med. Sci., 1904, cxxviii, 649.

<sup>4</sup> Ibid., 1909, cxxiii, 93.

<sup>5</sup> Ibid., cxxxvii, 186.

<sup>6</sup> Ibid., 1910, cxxxix, 870.

in three-fourths of all advanced cases of phthisis, although in his 171 cases of pulmonary tuberculosis a positive diagnosis of endocarditis was made only in 2. Similarly, N. B. Burns's<sup>1</sup> cases were diagnosed merely by the murmurs which were audible over the cardiac region, and he says that most of them were complications of phthisis.

In my own experience, I have seen but 5 or 6 cases of true mitral stenosis developing phthisis. To be sure, I have met with presystolic murmurs at the apex, but these murmurs, as well as the decompensation, appeared long after the onset of phthisis, mostly as a terminal phenomenon.

It seems that mitral stenosis causes a mechanical impediment to the lesser circulation, thus creating congestion or plethora of the blood-vessels in the lungs, and this has been offered as an explanation for the antagonism between this disease and phthisis. But it must be borne in mind that in compensated mitral stenosis the lungs do not have a larger quantity of blood than normally; it is only with the onset of decompensation that the pressure is elevated and the blood stream is slowed, thus favoring a larger quantity of blood in the lungs.

Those who accept the hematogenous origin of phthisis explain that in this manner the smaller vessels are dilated and the opportunity for development of emboli of tubercle bacilli is reduced to a minimum. In mitral stenosis the congestion of the pulmonary vessels is greater than in insufficiency, and for this reason phthisis is more rarely encountered in the former than in the latter.

In congenital heart disease, pulmonary stenosis appears to predispose to phthisis and those who survive infancy and childhood with such heart lesions, succumb during adolescence to tuberculosis because of the defective circulation of blood and lymph in the lungs which this cardiac defect brings about.

**Diabetes.**—For a long time diabetes has been considered as favoring the evolution of phthisis. It has been stated that the two diseases are very frequently associated and that phthisis in diabetics pursues a peculiar course, ending fatally in a short time. That glycosuria predisposes to tuberculosis has also been inferred from the fact that in animals the same condition has been observed. Thus, Schindelka reported pulmonary tuberculosis in a diabetic dog, and canines are usually very refractory to tuberculosis.

The first to collect statistics on the subject was Griesinger who, in 1859, reported 250 cases of diabetes in whom he found 42 per cent. affected with tuberculosis. Windle even found that 50 per cent. of 327 diabetics died from tuberculosis. But a more recent and thorough survey of the evidence by Charles M. Montgomery<sup>2</sup> shows that there is no conclusive proof that tuberculosis occurs more frequently in diabetics than in the general population at the same age periods. He found that out of 355 autopsies collected from the literature since

<sup>1</sup> Amer. Jour. Med. Sci., 1914, cxlvii, 866.

<sup>2</sup> Ibid., 1912, cxliv, 543.

1882, including his own 25 cases, 138, or 38.9 per cent., revealed pulmonary tuberculosis, mostly in an acute form. This cannot be said to be very excessive if we consider the frequency of tuberculosis in the general population at the ages between twenty and fifty.

It appears that diabetes hardly ever occurs in phthisical subjects. Whenever the two diseases are found in the same subject, the former was invariably the first disease. West, Raw, Montgomery, and others agree with this view. In my own experience, dealing with several thousand consumptives derived from a class (Jews) peculiarly predisposed to diabetes, I have never seen one developing glycosuria while suffering from active phthisis. The reasons for this peculiarity are obscure.

While in most cases tuberculosis occurring in diabetics runs a rapidly fatal course, which could be expected *a priori* considering that both are wasting diseases, I have seen many who lived on for many years. As Montgomery says, "Often each disease runs a course apparently independent of the other." We often see patients improving as regards their glycosuria or the pulmonary condition, or even both. I have a patient who has been diabetic and tuberculous for twelve years doing very well, excepting for occasional acute exacerbations of either condition.

**Acute Infectious Diseases.**—It has repeatedly been stated that the endemic contagious diseases, like measles, scarlet fever, whooping-cough, diphtheria, etc., are often followed by phthisis, and in infants and children tuberculous bronchopneumonia is frequently a sequel of measles and whooping-cough. This heightened predisposition may be explained as depending on the general disturbance in health caused by the fever, catarrh of the respiratory passages, etc., which reduce the resisting power and produce a soil favorable for the activation of dormant foci of tubercle bacilli, or favor new infections. The diseases are accompanied to a great extent by irritation of the mucous membranes and defects in the epithelium which facilitate the entrance of the bacilli, so that infection of the respiratory passages is particularly favored. The influence of measles and whooping-cough may be purely mechanical; fits of violent cough are liable to rupture tuberculous glands in the chest.

But E. P. Copeland<sup>1</sup> points out that "taking into consideration the enormous morbidity of measles, whooping-cough and influenza, the incidence of tuberculosis as a complication or sequel is of relatively small importance; that its development is all but invariably dependent upon the preëxistence of the latent disease; and that its dissemination is probably due to lymphatic activity resulting from the pulmonary inflammation associated with these diseases."

That these diseases may be strong predisposing factors to tuberculous infection and the extension of existing tuberculous disease, was

<sup>1</sup> Sixth Intern. Congr. Tuber., 1908, ii, 379.

shown from another viewpoint. "Allergy," or the altered reactivity of the organism to tuberculin, which is apparently dependent upon the fact that the body has produced antibodies which counteract the effects of tuberculous toxemia, is diminished in intensity or disappears altogether during an attack of measles. This "anergy" would indicate that resistance to infection has diminished, just as in far-advanced phthisis for a short period before the fatal termination, in miliary tuberculosis, etc., when all defensive powers have failed. Von Pirquet has named this state "anergic," *i. e.*, non-reacting. He assumes that the measles process occupies the antibodies which are needed for the repulsion of the tubercle bacilli present in the body. During this unprotected period the tubercle bacilli can grow and pass through the necrotic walls of a caseous gland, or secondary diseases can also occur, because now the circulating tubercle bacilli can find favorable conditions where at other times they would have been destroyed. He draws an analogy between this condition and the condition favoring the progress of tuberculosis in the adult—general debility due to malnutrition, overwork or any other condition robbing the body of its natural defences.

**Influenza.**—The connection between influenza and phthisis is even less clear. During the great pandemic of influenza in 1891 it was observed that the mortality was increased, and similar observations had been made before. It was therefore concluded that influenza is a strong predisposing factor in tuberculosis. But carefully studying the true conditions, we find that it was only the mortality from phthisis that was increased, and not the morbidity. Moreover, even this has not been lasting, for the mortality has been steadily declining despite the fact that influenza has been endemic all over the civilized world during the past thirty years. Clinically, we find that when a consumptive is subjected to an attack of influenza, the process in the lung is liable to extend, and acute exacerbation of the process is likely to occur which either kills the patient or turns a chronic and comparatively innocuous process into a subacute one, and finally to a fatal termination. We see this in hospital wards during epidemics of influenza; the mortality rises

**Typhoid Fever.**—Typhoid fever also has been considered as predisposing to phthisis because of the rather high proportion of consumptives who give a history of having passed through an attack of it. Recently, Charles E. Woodruff<sup>1</sup> has discussed the subject in great detail and arrived at the conclusion that typhoid fever heads the list of predisposing causes of tuberculosis. The fact that during recent years the mortality from tuberculosis and from typhoid has been declining at almost the same rate is considered a strong argument. "The three diseases which seem to be most frequently followed by tuberculosis of the lungs—measles, whooping-cough, and typhoid—are all complicated with bronchitis."

<sup>1</sup>American Medicine, N. S., 1914, xi, 17.

There appears to be a lack of evidence in support of these contentions. The fact that the mortality-rates from typhoid and phthisis run parallel does not prove that the same cause is operative in both cases. The somewhat excessive number of consumptives who have a history of typhoid does not convince in this direction. It is well known to clinicians that acute tuberculosis very often simulates typhoid in a striking manner, and with all our diagnostic methods it is often very difficult to differentiate the two diseases. In many cases of alleged typhoid preceding phthisis I have been convinced that it was an acute exacerbation of latent tuberculosis which was mistaken for typhoid, just as many attacks of "grippe" are in reality acute exacerbations of chronic or mild forms of phthisis. Typhoid fever, like most other febrile diseases, may, however, activate latent phthisis which might not have taken an acute or subacute course otherwise. But under the circumstances we cannot consider typhoid *per se* as predisposing to tuberculosis.

#### OCCUPATION.

Of the factors which have been mentioned as predisposing to the development and evolution of phthisis, the character of the occupation of the patient has been given prominence by nearly every writer on the subject. Very few, however, have looked at this problem with the view of William Gilman Thompson, who justly says that "it is often not the occupation which is at fault, but the manner in which it is conducted."

**Dust as an Etiological Factor in the Evolution of Phthisis.**—Long ago Ramazzini spoke of the etiology and relations of dust to diseases of the respiratory tract and at present, after we have studied the etiology of tuberculosis on a scientific basis, we find that the ancient clinician's observations have been confirmed in the main. In nearly all treatises on tuberculosis, or on occupational diseases, it is never omitted to state emphatically that persons pursuing occupations at which they are exposed to the inhalation of mineral, metallic, vegetable, or animal dust are more likely to contract tuberculosis and die from it than others. According to data obtained by the Twelfth Census of the United States the death-rate from phthisis was 5.41 per thousand among marble- and stone-cutters, as against only 1.12 per thousand among farmers and planters, and 1.07 among lumbermen and raftsmen. Statistics published by the United States Bureau of Labor in 1908-1909 show that the mortality from tuberculosis among males from twenty-five to thirty-four years of age constituted 31 per cent. of the total mortality in the working population. But among grinders it was 71 per cent.; among tool-makers, 59 per cent.; printers, 56 per cent.; stone-cutters and weavers, 55 per cent.; spinners, 50 per cent.; woolen-workers, 44 per cent. Similar statistics are available for many other countries, and for other occupations in which the workers are exposed to the inhalation of mineral

and metallic dust, especially grinders, tool- and instrument-makers, printers, etc. From a report of the Bureau of Labor in New York, it appears that the trades that showed the least effects from the ravages of consumption were the boot- and shoemakers, and millers.

It would seem that, with the exceptions to be mentioned later, mineral dust is the most dangerous in this regard, as has been shown by W. Zeuner,<sup>1</sup> Harlow Brooks,<sup>2</sup> Frederick Hoffman,<sup>3</sup> and others. Undoubtedly, it is the jagged and sharp-pointed particles which act as an irritant to the pulmonary tissues. Nature has placed many barriers in the way of even fine dust entering into the deep respiratory passages with the inspired air; even when reaching the mucous membrane of the bronchi and lung, the latter are very tolerant and most of it is soon expelled with the expectoration. But Moritz found that the sensibility of the respiratory tract from the nose to the trachea is reduced in persons working as grinders in a steel factory in Germany. Large masses of metallic dust could be seen lying on the vocal cords and mucous membrane of the trachea without provoking cough. For this reason some dust often remains and is taken up by the lymph channels and carried away. But after persistent deposits of dust in the alveoli, the irritation it produces excites a reactive inflammation, clogs up the lymph channels and lowers the resisting powers of the invaded lung, preparing the soil for the deposit of tubercle bacilli which may thrive in such defective areas of lung tissue. The glands of the lungs act as filters which retain the dust brought in by inhalation. But if new deposits of dust are brought repeatedly into these glands they are ultimately doomed to become damaged and their function as filters impaired, or even abolished. They are supersaturated with dust and, like a sponge which is supersaturated, can absorb no more. Zeuner is of the opinion that the glands of the deeper respiratory passages produce an internal secretion which is bactericidal, destroying any microorganism that may enter with the inspired air, including tubercle bacilli; at all events, it prevents their growth. Dust destroys the structure and function of these glands.

It appears that phthisis in patients with pneumokoniosis is often of a special form, pursuing a slow, sluggish course and with a symptomatology peculiarly its own. Fibroid phthisis, which will be discussed later on, is mostly found in workers exposed to the inhalation of animal or vegetable dust. The foreign particles deposited in the alveoli excite a productive inflammation. At first, small diseased foci are produced, but later, if the irritation keeps up, the small foci coalesce, affecting extensive areas of pulmonary tissue, and tubercle bacilli, either brought by the inspired air, or by metastatic deposits from old, latent lesions, invade these areas secondarily.

<sup>1</sup> Luftereinheit zur Bekämpfung der Tuberkulose, Berlin, 1903.

<sup>2</sup> Dietetic and Hygienic Gazette, 1907, xxiii, 709.

<sup>3</sup> Bull. Bureau of Labor, November, 1908, p. 633.

This form of phthisis may last for years without greatly incapacitating the patient, who may have no fever, no debility, no night sweats, etc.; only cough and expectoration, and very often dyspnea, being the annoying features clinically. I have observed this form of phthisis among garment-workers, notably furriers, in New York City.

"In New York City," says William Gilman Thompson,<sup>1</sup> "a hotbed of tuberculosis is found in the so-called 'sweat-shops' where so much ready-made clothing is manufactured. If a man comes to my Cornell Out-patient Clinic and gives his occupation as 'tailor's presser,' I always ask him at once how long he has had a cough. He is almost certain to have worked in a densely crowded, unventilated room, dusty from the lint of clothing, and with his tailor's iron heated over a gas stove, which adds to the vitiation of the atmosphere. He has had long hours of work and poor food. Thus, anemic, ill-nourished and fatigued, his body is in ideal condition for the development of the germs of tuberculosis."

But not all dust is etiologically related to phthisis. Thus, among coal-miners, who undoubtedly inhale large quantities of mineral dust, as is evident from the frequency of pneumokoniosis among them, true tuberculous phthisis is comparatively infrequent. Kuban drew attention to this fact as far back as 1863 in France where "coal dust is unable to cause pulmonary tuberculosis or even favor the evolution of pulmonary tubercle. It prevents the development of phthisis." In his book on occupational diseases, Oliver shows that this is true of English coal-miners, and in the United States Wainwright and Nichols<sup>2</sup> found that in Scranton, Pa., tuberculosis is about two-thirds less frequent among miners than among all other occupied males. Some writers have attempted to explain this paradox by assuming that coal dust possesses antiseptic properties, and is rather a protection against tuberculosis. Cornet suggests that in coal mines the air is humid and thus prevents desiccation and pulverization of sputum which is, of course, far-fetched.

More noteworthy is it that street-sweepers and coachmen, in spite of exposure to excessive inhalation of dust, are not excessively liable to phthisis. Cornet concludes from this fact that the dangers of infection in the street are *nil*. Sommerfeld has shown that in Berlin the street-sweepers have only half the rate of mortality from phthisis when compared with the mortality of the working classes in that city. In New York City, where several years ago considerable agitation was made in favor of protecting the street-sweepers against the excessive morbidity and mortality from tuberculosis, statistics have not borne out these contentions. Hoffman's<sup>3</sup> statistics, gathered for a monograph on the excessive mortality from consumption in occupations exposed to municipal and general dust, show that evidently "the recorded

<sup>1</sup> The Occupational Diseases, New York, 1914, p. 58.

<sup>2</sup> Amer. Jour. Med. Sci., 1905, cxxx, 405.

<sup>3</sup> Bull. Bureau of Labor, November, 1908, p. 633.



mortality from consumption among men in this employment is not decidedly excessive."

Another kind of dust which is harmless in this regard is limestone and also plaster of Paris. In England it has been found, according to Edgar L. Collis,<sup>1</sup> that masons in districts where limestone is worked do not suffer from phthisis in excess; while masons in districts where sandstone is worked are peculiarly liable to succumb to this disease, and have a shorter prospect of life. Halter and Garb have observed the same to be the case in Germany, and G. Fisac<sup>2</sup> reports that in Spain the workers in quicklime and plaster of Paris are immune to tuberculosis despite the fact that they live in squalid dwellings and are underfed. He believes that their immunity is due to the inhalation of dust containing lime.

That the chemical composition of the dust is of more importance than the dust itself is well shown by Collis in his *Milroy Lectures* for 1915. He finds that when phthisis occurs as a result of inhalation of mineral dust, it is always associated with exposure to dust containing crystalline silica, though he could find no definite relation between the amount of dust present and the prevalence of phthisis. As to why coal dust, lime, plaster of Paris, etc., should be harmless in this regard, while flint, slate, iron, tin, lead, etc., do produce pulmonary tuberculosis, we are at a loss, and it may be worthy of further study.

Another point brought out by Collis is that phthisis encountered among workers at dusty occupations is actually due to the inhalation of the dust, and not to their mode of life. Outdoor workers inhaling dangerous dust succumb, while careless indoor workers at dusty occupations inhaling dust containing no silica, or metallic fragments, are not excessively liable to phthisis. He finds that "dust phthisis is peculiar in showing a low degree of infectivity among contacts not exposed to dust inhalation." In the lead-mining districts of England, there is a larger proportion of widows than in any other place in the kingdom. Haldane observed among tin-miners that "the wives and children of these men never seem to be affected, although occupying the same room as the affected men who never go to the hospital, but sit at home and expectorate sputum loaded with tubercle bacilli." Barwise noted the same phenomenon among gritstone-workers in Derbyshire, and it is also true of stone-masons according to Collis.

This shows clearly that certain forms of dust are capable of waking up dormant tuberculous lesions in the workers; but their wives, who have assuredly been infected with tubercle during childhood, cannot be reinfected with the bacilli expectorated by their husbands. It entirely agrees with our modern views of tuberculous infection, and the difficulty or impossibility of reinfection which is spoken of in Chapter V.

It thus appears that occupation *per se* cannot be considered as pre-

<sup>1</sup> Public Health, 1915, xxviii, 252, 292; xxix, 11.

<sup>2</sup> Rev. de hig. y de tub., 1909, v, No. 54.

disposing to phthisis, with the exception of those which involve exposure to metallic and certain kinds of mineral dust. But even in these there are exceptions, as we saw, with street dust, coal dust, limestone, plaster of Paris, etc. Thus, there has been found a relation between the wages paid to workmen and the incidence of phthisis among them. B. S. Warren's<sup>1</sup> study of conditions in the United States Government printing and engraving plants shows that despite the fact that they are badly overcrowded, with poor ventilation, etc., the mortality from tuberculosis is rather low among the employees. The reason he assigns is that they receive good wages. He finds from census statistics that low wages go hand-in-hand with a high tuberculosis mortality. The difference in wages or income means a difference in nutrition, social contentment, and general welfare which renders the farm laborer more susceptible to phthisis than his employer, and the cotton-mill operative more than the general population. Similarly, he finds that of deaths among males reported by the Census Bureau for 1909, giving the occupation of the deceased, 14.7 per cent. were from tuberculosis, as against 20.9 per cent. among females. The reasons for this disparity are many, but undoubtedly the inadequate wages paid to women are responsible for a considerable portion of the phthisis among female workers.

**Injury as a Cause of Phthisis.**—That traumatism may determine the localization of extrathoracic tuberculosis—of the bones, joints, glands and meninges—is a well-known and accepted clinical fact supported by the results of animal experimentation. But that a local injury to the chest may be the exciting cause of phthisis is not generally appreciated to the extent it deserves. It seems that the older medical literature only rarely referred to this subject, and Grasser could only find reports of about 50 cases before 1903. In the Prussian Army it was observed that among 6924 cases of phthisis, 95 began after an injury, and of these 79 had sustained contusions of the chest. This would indicate that it is more frequent than was formerly appreciated.

It is obvious that an injury *per se* cannot cause tuberculosis of a bone or a joint. Tubercle bacilli must be present. But in the light of our present knowledge of phthisiogenesis it is clear that many, if not most, persons harbor some latent or healed tuberculous foci with virulent tubercle bacilli which an injury may reawaken into activity. Külbs has shown that contusions of the chest often cause lacerations and hemorrhages of the pulmonary parenchyma, even when no visible hemoptysis occurs, and such lacerated areas may offer a favorable soil for the implantation of tubercle bacilli, just as a wound in a joint or a fractured rib.

In his monograph on this subject Richard Stern<sup>2</sup> gives the following direct and indirect possibilities of phthisis after injury: (1) A peripheral tuberculosis of a bone or joint may be produced and this may

<sup>1</sup> Trans. Nat. Assn. Study and Prev. Tub., 1913, ix, 153.

<sup>2</sup> Die traumatische Entstehung innerer Krankheiten, Jena, 1910.

influence unfavorably a preëxisting tuberculous lesion in the lung; (2) the unfavorable influence of loss of blood; (3) peripheral thrombosis may be caused, followed by pulmonary infarction which may ultimately end in secondary tuberculous infection; (4) the deleterious effects of a long stay in bed, especially in hospitals; (5) psychic depression, reducing the general resisting powers and producing changes in the constitution of the patient as a result of the accident.

In persons known to be tuberculous the disease may be aggravated by an injury, as I have seen in several cases, and lead to a fatal termination. Especially is this the case when hemoptysis is caused by the injury. Traumatism may also produce pleurisy, usually dry, but occasionally with an effusion. Pneumothorax is another possible result of an injury to the chest. In non-tuberculous traumatic pneumothorax the rent in the pleura heals quickly and the air is absorbed, but in those with a preëxisting tuberculous lesion in the lung, active or dormant, the usual course of spontaneous pneumothorax, hydrothorax, pyopneumothorax, etc., may be followed.

The intensity of the injury should not be taken as a measure of the probability of its relation to phthisis subsequently developed, as has been pointed out by Wolff-Eisner. After violent injuries to bones, especially those resulting in fractures, tuberculous osteomyelitis is hardly ever observed, though slight injuries to bones may be followed by local tuberculosis. In the same manner, as I have seen in several cases, a slight injury to the chest may flare up a latent tuberculous process. In persons known to be healthy this is not uncommon. John B. Hawes<sup>1</sup> points out that after the autumn football season some players develop consumption as a result of injuries received on the football field. The special diet usually prescribed by the trainer, as well as the excessive exertion for months during the training period, undoubtedly reduces the resisting powers of even gridiron heroes.

The site of the lesion provoked by an injury is not necessarily at the point affected by the blow. Many authors have reported lesions by *contrecoup*. An acute general or miliary tuberculosis may also result from breaking up of a latent lesion and letting loose tubercle bacilli into the blood stream. Hemoptysis is not absolutely essential to establish the relationship between the injury and the phthisis, because laceration of the lung may occur without causing hemorrhage. When hemoptysis occurs, the quantity of blood expelled is no criterion of the size of the torn vessel. Nor must there remain any external marks on the chest wall because an injury may lacerate the lung or pleura without leaving any external traces.

The appearance of clinical symptoms of phthisis may be delayed for some time. Of course, in cases of quiescent lesions which are activated as a result of traumatism, the aggravation in the condition of the patient, and the extension of the process, may appear soon after

<sup>1</sup> Boston Med. and Surg. Jour., 1913, clxviii, 83.

the accident, and hemoptysis may appear even immediately. But in apparently healthy persons the symptoms may appear many months or years later. Hawes reports several cases in which phthisis developed from two to ten years after the injury.

It takes about eight weeks for a tubercle to develop and one tubercle is by far not enough to give symptoms or signs by which it can be recognized by the patient or the physician. In fact, when a few days after an injury signs of phthisis are found, especially tubercle bacilli are found in the sputum, we may conclude that we are dealing with a preëxisting disease which was, at most, aggravated by the accident. But in cases in which the symptoms, such as fever, emaciation, cough, expectoration, etc. make their appearance three to six months after the injury in a person known to have been well before the accident, and the physical signs appear even later, it is clear that there was a causative relation between the injury and the disease. German authorities have limited the time for the appearance of the symptoms after the injury to six months, although there are undoubtedly exceptions which must be judged on their individual merits.

## CHAPTER V.

### PHTHISIOGENESIS.

**Tuberculosis vs. Phthisis.**—After infecting an animal with tubercle bacilli, we know exactly what morbid phenomena to expect. On injecting into the peritoneal cavity of a guinea-pig a certain quantity of the pure culture of tubercle bacilli, tuberculous peritonitis soon develops, followed by tuberculosis of other organs—the spleen, the liver, the kidneys, etc., until it finally succumbs. But what will happen after a human being is infected in the usual spontaneous manner we cannot prognosticate with any degree of certainty. The individual may pass through life without showing any morbid manifestations which can be attributed to the infection. In fact, the vast majority of people have been infected during their childhood and are none the worse for their experience, as has already been shown. A large proportion of those in whom distinct lesions of a tuberculous character have been found at the autopsy knew nothing about it during their life. On the other hand, in a certain proportion the infection is followed sooner or later by some clinical form of tuberculosis.

This is, however, not the only difference between experimental tuberculosis and spontaneous phthisis as we meet it in human beings.

It appears that phthisis is a disease met with exclusively in human beings and rarely, if ever, in the lower animals; certainly not in animals which have been infected experimentally in the laboratory, be it by inoculation, ingestion or inhalation of tubercle bacilli. In guinea-pigs, rabbits, etc., in whom spontaneous tuberculosis is exceedingly rare, only nodular tubercles consisting of avascular, cellular masses are formed after experimental infection; while spontaneous human phthisis is mainly a productive and exudative inflammatory process of the lungs in which there may or may not be any of the characteristic tuberculous cell-proliferation. In other words, in animals it is general or miliary tuberculosis that we find, and this is also rarely met with in humans. "Real pulmonary tuberculosis" says von Hansemann,<sup>1</sup> "in the anatomical sense is always part and parcel of general tuberculosis of all the organs in the body. Pure and isolated pulmonary tuberculosis in the anatomical sense, *i. e.*, in which there are no other tuberculous changes in the lungs than the development of submiliary tubercles, never occurs as far as my experience goes. But it is a noteworthy fact that from this disease, which in reality alone deserves the name pulmonary tuberculosis, phthisis never

<sup>1</sup> Berliner klin. Wehnschr., 1911, xlviii, 1.

evolves. I know of no case in my own experience, nor from medical literature, in which the disease began as acute miliary tuberculosis in the anatomical sense, and then turned into pulmonary phthisis." But phthisis may be complicated by general miliary tuberculosis. This often occurs before the fatal termination of the case.

In the same sense we find that Ribbert<sup>1</sup> makes a sharp distinction between experimental tuberculosis in animals and phthisis in human beings: "It is undoubtedly a fact that tubercles may be produced in the lungs of animals which are made to inhale dust containing tubercle bacilli. But, (1) the disease thus produced is not the same as that in human beings; (2) we cannot, without further proof, conclude that human beings are infected in the same manner. The conditions under which humans inhale tubercle bacilli are, at least from the viewpoint of quantity, distinctly different from those prevailing during experimentation. It can neither be proved that individuals always inhaled tubercle bacilli before becoming sick, nor that the latter settled primarily in the particular organ in which they proliferated. Neither the clinical nor the anatomical findings sufficiently support such a view. It is self-understood that I do not in the least deny that in man also disease may directly follow the inhalation of tubercle bacilli, but it is a question how often this takes place. From mere possibility to uncontrovertible proof which will cover all tuberculous diseases of the lungs, is quite a distance." "Pulmonary phthisis" says Bacmeister,<sup>2</sup> "is a disease found exclusively in adult human beings; it never occurs spontaneously in animals, nor has it ever been produced experimentally."

If we want to apply unequivocally the experimental findings to man we must first demand that infection of animals should result in isolated apical lesions which should extend gradually downward in the lung in the typical chronic manner. All other forms of tuberculosis which are produced experimentally in the lungs of animals do not prove much, because their morbid anatomy diverges so much from the changes found in human phthisis.

The problem why the human adult after infection with tubercle bacilli develops phthisis, a disease unknown in early childhood and among the lower animals, has not yet been solved to the satisfaction of all who are entitled to an opinion. Freund, Hart, Bacmeister, and others believe that pressure of a short rib or an ossified first costal cartilage upon the apex of the lung is responsible for the apical localization of phthisis (see p. 84). We have, however, shown that this theory does not explain everything connected with the problem. Various other theories have been promulgated to explain the origin of human phthisis.

**Phthisis as a Disease Acquired during Childhood.**—During recent years the theory that phthisis is a late manifestation of tuberculosis

<sup>1</sup> Die Ausbreitung der Tuberkulose im Körper, Marburg, 1900.

<sup>2</sup> Die Entstehung der menschlichen Lungenphthise, Berlin, 1914, p. 35.

acquired during childhood has been gaining ground. Behring,<sup>1</sup> basing his opinions on experiments with guinea-pigs, maintains that a single infection cannot result in phthisis. He says that phthisis is the result of reinfection of a person who was already once infected during infancy, mainly through deglutition of milk derived from tuberculous cows. The bacilli pass through the gastro-intestinal tract into the lymphatics where they remain for years in an avirulent or mildly virulent state, and in the adult, as a result of some intercurrent affection, they become again virulent and cause phthisis. "Phthisis is but the last verse of the song, the first verse of which was sung to the infant at its cradle."<sup>2</sup>

Hamburger's<sup>3</sup> conception of phthisis is also that it must not necessarily be preceded by recent infection, but that it is rather a reawakening or an exacerbation of an old, "healed," or latent tuberculous process. He points out that tuberculosis runs a different course in children than in adults—pulmonary phthisis which is so frequent in adults is exceedingly rare in children. But we know that most people have passed through a tuberculous infection during childhood. Under the circumstances the inference is justified that pulmonary phthisis is invariably preceded by a tuberculous infection many years before its onset.

To Hamburger the course of phthisis is similar to that of syphilis, with periods of health and quiescence or latency, interrupted or followed by periods of acute or subacute exacerbations. The primary lesion is inoculated during childhood, before the individual reaches his tenth year of life. During infancy this primary focus, if massive infection has taken place or the resistance is low, may cause miliary tuberculosis, or hematogenic metastasis, but in the vast majority of people it heals or remains dormant. In those in whom metastatic deposits of tubercle bacilli are distributed in various parts of the body, secondary tuberculous manifestations make their appearance, consisting in tuberculosis of the glands, bones, joints, meninges, etc. After the tenth year the tertiary manifestations are met with, consisting in the various forms of chronic pulmonary phthisis, tuberculosis of the larynx, tumor albus, certain cases of joint diseases, of the kidneys, lupus vulgaris, tuberculous iritis, adhesive pleurisy, etc. These last are practically never seen in infancy and early childhood; only after the disease has lasted for many years they may appear, just as the late manifestations of syphilis—tabes, general paralysis, etc., are only rarely seen in early youth, although syphilis is quite frequent at that period of life.

Phthisis is thus, according to Hamburger, an exacerbation of tuberculosis which has been acquired during early childhood and remained

<sup>1</sup> Deut. med. Wehnschr., 1903, xxix, 689; Brit. Med. Jour., 1903, ii, 993.

<sup>2</sup> Einführung in die Lehre von der Bekämpfung der Infektionskrankheiten, Berlin, 1912, p. 354.

<sup>3</sup> Die Tuberkulose des Kindesalter, Leipzig, 1912.

latent for many years until some exciting cause, or a reduction in the powers of resistance, has brought about conditions favorable for its development.

**Immunity or Allergy.**—The view of phthisiogenesis which has been gaining ground of late, and which is apparently based on a sound foundation, has been formulated by Paul Römer to the effect that *phthisis is a manifestation of immunity against tuberculosis which has been acquired by an infection during early childhood.*

It appears that the observations made in most of the transmissible diseases that one attack renders the individual immune against renewed infection with the same virus, hold good in tuberculosis. Behring, Römer, Calmette, Metchnikoff, Hamburger, and many others have shown that the mild infections with tuberculosis during childhood endow the organism with a certain amount of immunity against further and renewed exogenic infection with tubercle bacilli, so that an individual with a healed or latent lesion, acquired during early childhood, is immune to these microorganisms. Repeated infection with the same virus may be reinfection or superinfection. By superinfection is understood a second infection at a time when the lesions produced by the first infection have not healed, while reinfection implies a new infection when the lesions produced by the first have completely healed. "Inasmuch as we may accept as a great probability that in tuberculosis healing in the strict scientific sense never occurs," says Hamburger,<sup>1</sup> "all repeated infections in tuberculosis are to be considered superinfections." We use the word *reinfection* because this term has gained extensive currency in medical literature.

**Experimental Proofs of Immunity.**—Experimentally acquired immunity by an inoculation of tuberculosis has been proved to exist by the researches of Koch,<sup>2</sup> Behring, Römer,<sup>3</sup> Hamburger, Webb and Williams,<sup>4</sup> Rossignol, Krause and Volk, and many others. When a healthy guinea-pig is inoculated with tubercle bacilli in pure culture, the wound closes up within a couple of days and seemingly heals up. But about ten to fourteen days later there appears at the site of the inoculation a hard nodule which soon breaks down, leaving an ulcer which persists till the animal dies. It is different when a tuberculous animal is inoculated with tubercle bacilli. The wound also heals, but no nodule is formed and a few days later the point of inoculation becomes indurated, dark in color all around the punctured point to about 1 cm. in diameter. During the next few days the spot becomes necrotic and the involved tissues are shed, leaving a flat ulcerated area which usually heals quickly and permanently. Moreover, while after infecting a healthy animal the regional lymph glands become swollen, this does not occur after reinfection of a tuberculous animal.

<sup>1</sup> Med. Klinik, 1915, xi, 34.

<sup>2</sup> Deutsche med. Wehnschr., 1891, xvii, 101.

<sup>3</sup> Beitr. z. Klinik d. Tuberkulose, 1910, xvii, 287; 1912, xxii, 301.

<sup>4</sup> Jour. Med. Research, 1911, xxiv, 1.



The work of Römer<sup>1</sup> and Hamburger<sup>2</sup> along these lines has recently changed our conception of tuberculous infection and suggested prophylactic measures which are actually revolutionary. They have found that reinfection is as difficult and even as impossible in tuberculosis as in syphilis. All modes of infection were tried, inoculation, feeding and inhalation of tubercle bacilli in dust or spray, and contact infection, which are akin to the usual modes of spontaneous infection in human beings, but no new tuberculous lesion could be produced in tuberculous animals, while the healthy controls were infected and succumbed to the disease in some form. Not only were guinea-pigs and rabbits—which are very susceptible—thus tried, but sheep which are not as vulnerable to tubercle bacilli, and also dogs which are strongly refractory, and monkeys which display the same degree of susceptibility as man. Römer found that when a healthy sheep is infected with a certain dose of tubercle bacilli, it succumbs within eight weeks to acute pulmonary tuberculosis, but the same dose is harmless in a tuberculous sheep. In monkeys the results were the same. Hamburger and Toyofuko have proved that infected guinea-pigs are not only immune to inoculation but also to inhalation which is deadly to healthy control animals. It appears from Römer's studies that this immunity is not transmitted by heredity, even when displayed by pregnant mothers.

It has also been found that this immunity is not only true of exogenic superinfection, or additional infection with bacilli of another strain, but also of superinfection with bacilli taken from their own lesions.

Another important point was established by the experimental investigations of Römer and Hamburger: If the reinfecting dose of tubercle bacilli is small, perfect immunity is found—the point of inoculation heals quite soon. As a rule, the immunity is observed in animals which have been tuberculous for some time, three or four months. But if the reinfecting dose of tubercle bacilli is massive, it soon causes death of the animal.

These experimental researches are well founded, having been confirmed by many workers in various countries, so that at present they are as firmly established as anything else we know about spontaneous and experimental tuberculous infection, but there arise several problems of immense interest in our study of phthisiogenesis. Knowing well that the vast majority of human beings have been infected with tubercle bacilli during childhood, even those who have no clinical evidence of phthisis, we may justly ask, Can adults be infected with tuberculosis at all? The bearings of this problem on prophylaxis are enormous. How does phthisis develop from the lesions acquired during infancy and childhood? Is it due to a second infection immediately before the onset of the disease, or do the old, hitherto dormant lesions for some reason flare up and begin to extend?

<sup>1</sup> Beitr. z. Klinik d. Tuberkulose, 1910, xvii, 287, 383; 1912, xxii, 265, 301.

<sup>2</sup> Ibid., 1910, xvi, 271.

**Modés of Reinfection in Human Beings.**—A person who has once been infected with tubercle bacilli may be reinfected with the germs which he harbors within his body, or with bacilli which have grown in the body of some other person or in an animal. In the case of endogenic or autogenic reinfection the process may be very simple: A softened tuberculous lesion in the lung is ruptured into a bronchus, and during cough the tuberculous material is carried along the bronchial tree to some other part of the lung where it is deposited and, taking root, it produces a new lesion. In this manner there may also be produced laryngeal and intestinal tuberculosis, the latter from swallowed sputum. But endogenic reinfection is not always bronchogenic; it may also be hematogenic—a tuberculous lesion may break into a bloodvessel and then bacilli are carried to various parts of the body; or it may be lymphogenic, the tuberculous material is carried by the lymphatics, infecting the lymph glands, etc.

Exogenic reinfection should be very common, if it takes place at all. The bacilli are ubiquitous, and one suffering from any form of tuberculosis is evidently predisposed, otherwise he would have escaped the disease, despite the first infection. Infection is exceedingly easy, as is evident from the fact that when a child free from tuberculosis is brought in contact with a consumptive, it is soon infected. Hamburger even reports a case where exposure of an infant for one hour was effective in infecting it. We also see this to be a fact in adults: When individuals free from tuberculous infection dating back to childhood, as is the case with primitive peoples, come into contact with tuberculous people, they are soon infected and succumb in a short time.

Granting these premises, which are based on carefully observed facts, we may be able to study the problem of reinfection in man clinically, even though the experimental method is, for obvious reasons, closed to us. All we have to do is inquire into the frequency of exogenic and endogenic superinfection and reinfection in tuberculous patients who are inmates in hospitals for consumptives; the frequency of tuberculosis among those who are apparently healthy but live with consumptives; and also the effects of tuberculous infection on persons who are known to have escaped infection during childhood.

**Reinfection in Hospitals for Consumptives.**—Clinical experience has shown that it is one of the rarest things in medicine that a person should have one of the exanthemata twice during his life. It has also been observed that in a ward filled with cases of scarlet fever, smallpox, etc., there is no danger that patients suffering from the more malignant types of the disease should transmit the virus to those who are passing through a mild or abortive attack of the same disease. In nearly all contagious and infectious diseases we find that during the existence of the malady the patient is immune against exogenic reinfection with the virus of the same disease. The same is true of the exceedingly chronic transmissible disease, syphilis.

The experience in hospitals harboring large numbers of consumptives

should give us information along these lines about tuberculosis. Here the patients have all the opportunities for superinfection with bacilli derived from other patients. For it must be agreed that despite the scrupulous cleanliness observed at present in sanatoriums and hospitals, it is impossible to avoid droplet infection when many patients are brought into intimate contact. In fact, when caged guinea-pigs are kept in scrupulously clean wards they soon contract tuberculosis.

It has, however, never been observed that a mildly infected patient living in an institution should be reinfected from one severely infected who shares the ward with him, even when the latter expectorates myriads of virulent bacilli and offers exceptional opportunities for droplet infection.

Many non-tuberculous patients remain in sanatoriums for months, yet it has not been observed that one should become tuberculous because of his sojourn in the hospital. This is the reason why hospitals and sanatoriums do not separate the "open" from the "closed" cases, *i. e.*, those who expectorate sputum reeking with tubercle bacilli from those who do not, in spite of the fact that many physicians are convinced that droplet infection is a potent factor in disseminating tuberculosis.

The hospital staff, including physicians, especially laryngologists, nurses, orderlies, etc., come in close contact with the patients in sanatoriums and should become infected if adults, presumably infected during childhood, could be reinfected with tubercle bacilli. But, if experience of thousands of people in these callings counts for anything, they do not show a higher mortality nor morbidity from tuberculosis than persons in other occupations. The first statistics bearing on this problem were published by C. Theodore Williams<sup>1</sup> who showed that long before the discovery of the tubercle bacillus, and before any precautions were taken to prevent the transmission of the disease, no case of infection of the hospital staff had been observed. From 1846, when the Brompton Hospital for Consumptives was opened in London, to 1882 "statistics showed that among the physicians, assistant physicians, hospital clerks, nurses and others, to the number of several hundred, who had served in the hospital (not few of them having lived in it for a number of years continuously), phthisis had not been more common than it may be expected to be on the average among the civil population of the town." In a later paper Williams<sup>2</sup> brought these statistics down to 1909 and found that conditions remained the same. But while during recent years the improvements in hygienic conditions and disinfection of sputum may be the cause of the rarity of phthisis in the hospital staff, this cannot be said to have been operative before 1882.

Similar statistics are available for hospitals in Germany and France,

<sup>1</sup> Brit. Med. Jour., 1882, p. 618.

<sup>2</sup> Ibid., 1909, ii, 433.

published by Aufrecht,<sup>1</sup> Freymuth,<sup>2</sup> Brunon,<sup>3</sup> Saugman,<sup>4</sup> and others, and brought together by the author<sup>5</sup> in a paper on hospital infection. Instructive data on the subject have been collected by Saugman from many sanatoriums in various countries. He finds that even among laryngologists, who are exposed to infection more than any other class, the morbidity and mortality from tuberculosis is less than would be expected. He concludes that tuberculosis is extremely rare among those who are engaged among consumptives; physicians and laryngologists who had been healthy before entering upon their duties, remain so. "It is not dangerous for healthy adults to be coughed at by patients suffering from pulmonary or laryngeal tuberculosis" concludes Saugman.

Such facts have been quoted to disprove the transmissibility of tuberculosis, but in the light of our present knowledge they merely prove that reinfection is impossible.

**Marital Phthisis.**—Again, bearing in mind the ease with which tuberculosis is transmitted to persons who have not been infected previously, it should be expected that the vast majority of husbands of tuberculous wives, or healthy wives of tuberculous husbands should acquire the disease. This, we know, is the case with syphilis, in which the active disease is almost invariably transmitted to the unaffected consort, excepting when the latter has been infected before marriage. But for a long time it has been a mystery why phthisis in both husband and wife is very rare in spite of the fact that they probably come into more intimate contact than even father and child. Even in families in which most, or all, the children are affected with tuberculosis it is exceedingly rare to find that both the mother and father should be sick with the disease. Formerly this fact was used as a strong argument against the transmissibility of tuberculosis, but now we understand that it is due to the immunity acquired by an infection which has not been effective in producing phthisis.

For many years the writer was physician to a charitable society, having under his care annually 800 to 1000 consumptives who lived in poverty and in want, in overcrowded tenements, having all opportunities to infect their consorts; in fact most of the consumptives shared their bed with their healthy consorts. Still, very few cases were met in which tuberculosis was found in both the husband and the wife. Widows, whose husbands died from phthisis, were only rarely seen to develop the disease.

This experience is not unique. Mongour<sup>6</sup> found that among 440 married couples, in which one of the consorts was sick with tuberculosis, there were only 16 in which the partner was also phthisical, *i. e.*, 4 per

<sup>1</sup> Münch. med. Wchnschr., 1908, xlv, 158.

<sup>2</sup> Beitr. z. Klinik d. Tuberkulose, 1911, xx, 231.

<sup>3</sup> La tuberculose pulmonaire, Paris, 1913, p. 59.

<sup>4</sup> Zeitschr. f. Tuberkulose, 1905, vi, 125; 1907, x, 224.

<sup>5</sup> American Medicine, 1915, xxi, 607.

<sup>6</sup> Cong. Intern. de la Tuberculose, Paris, 1905, i, 413.

cent. Thom<sup>1</sup> reports of 402 couples with only 12, or 3 per cent., in which infection of the consort had taken place in all probabilities. I. Burney Yeo<sup>2</sup> found marital phthisis comparatively rare, basing his deductions on particulars collected of 1055 cases of consumption. He cites figures of J. R. Bartlett, Herman Weber, and others and concludes: "Taking these figures for what they are worth, it seems certain that the communication of consumption from wife to husband, even among the class in which the conditions of life favor to the utmost the communication of contagious disease, is very rare; while it would seem that communication from husband to wife is more frequent." Pope,<sup>3</sup> Pearson,<sup>4</sup> Elderton, and Goring have made careful statistical studies of this problem in England and arrive at the conclusion that the chances of tuberculosis occurring in both consorts are about the same as insanity, and a German writer has shown that cancer in both consorts is more apt to occur than phthisis. In a recent statistical study by Levy,<sup>5</sup> comprising 317 married couples which lived in poverty, 34 per cent. sharing the bed, possible marital infection could be traced only in 2.8 per cent. He points out that when marital phthisis does occur, it is characterized by a favorable course of the disease in the secondary cases, and soon after the actively diseased partner is removed, the infected consort recovers his or her health. Haupt found among 1553 tuberculous couples that 106, or 7 per cent., were both affected. This being the highest percentage recorded, it is essential to remember that it is exactly the proportion in which humanity suffers from the disease.

Römer mentions that life insurance companies in Germany, basing their action on statistical experience, do not reject persons because of a history of exposure to infection, or those who live with tuberculous consorts. George Florschütz<sup>6</sup> in his work on insurance selection, says that "in medical selection one must certainly consider the risk of infection when it is so evident as in conjugal intercourse, but in general as far as life insurance is concerned, one may regard tuberculous infection as purely a matter of chance." He brings statistics "showing that of 1428 deaths from tuberculosis, there were but 11 in which the husband or wife of the deceased were tuberculous."

We have dwelt on these facts because they are very important points in phthisiogenesis: (1) tuberculous infection can only occur once; and (2) that phthisis evolves only in persons who are for one reason or another predisposed to the disease. Inasmuch as the non-phthisical consort has already been infected with tubercle bacilli during childhood, all new opportunities for reinfection by cohabitation with a

<sup>1</sup> Zeitschr. f. Tuberkulose, 1905, vii, 12.

<sup>2</sup> Brit. Med. Jour., June 17, 1882, p. 895.

<sup>3</sup> A Second Study of Statist. of Pul. Tuberc. Marital Infection, London, 1911.

<sup>4</sup> Tuberculosis, Heredity and Environment, London, 1912; The Fight against Tuberculosis and the Death Rate from Phthisis, London, 1911.

<sup>5</sup> Beitr. z. Klinik d. Tuberkulose, 1914, xxxii, 147.

<sup>6</sup> Medical Record, 1915, lxxxvii, 957.

consumptive consort are of no avail to produce phthisis. It is his or her constitution that determines whether consumption will develop, and not the opportunities for reinfection.

**Clinical Proofs of Immunity Acquired by Tuberculous Infection.—**

Many investigators have shown that tubercle bacilli circulate in the blood of a large proportion of consumptives, yet they do not manifest general or miliary tuberculosis, as would *a priori* be expected. The only plausible explanation is that inasmuch as they have already a tuberculous focus in some part of the body, this protects them against renewed endogenic or exogenic reinfection, and the bacilli in the blood remain innocuous.

A number of clinical facts, hitherto obscure, can be explained by this acquired immunity of the tuberculous to tuberculosis and they confirm the assumption that the experimental data obtained in animals hold good for man. Thus, in spite of the fact that so much sputum containing tubercle bacilli, passes through the throat, tonsils, mouth, lips, etc., tuberculosis of these mucous membranes and the cervical glands is exceedingly rare in adults. Conversely, in former times physicians believed that scrofulous children were immune to phthisis, and my observations leads me to the conviction that this is true today.

Calmette<sup>1</sup> says: "Everyone knows that a local tuberculous suppuration occurring in a person with pulmonary tuberculosis ameliorates the condition of the patient and considerably increases his resistance. Inversely, it is rare that patients in whom pulmonary tuberculosis has had a rapid development have been attacked previously by supuration of the lymph nodes, or bony or cutaneous tissues, except in cases where an inopportune surgical operation has provoked infection of the blood. It is a well-known fact that about a quarter of the persons suffering from lupus present the auscultatory signs characteristic of pulmonary tuberculosis, and that these generally develop in them with very great slowness; likewise many lupus patients live to advanced age." Marfan also found that persons with healed tuberculosis of the skin and glands never become phthisical, and Piéry<sup>2</sup> shows that a certain number of children of tuberculous parentage display a veritable immunity against the grave and acute forms of tuberculosis. They are just the ones who present the alleged stigmata of tuberculous heredity, which predisposes according to some authors.

Mayo<sup>3</sup> pointed out that in Minnesota, where surgical tuberculosis is rife, phthisis is uncommon, and this has been observed to be a fact in other places. Turban, Weicher, and King record the more favorable course of phthisis where a family history of tuberculosis is present and the same is the case where the individual has been scrofulous. Clive Riviere is inclined to attribute the scrofulous manifestations,

<sup>1</sup> Medical Record, 1908, lxxiv, 741.

<sup>2</sup> Lyon Médicale, 1910, cxv, 889.

<sup>3</sup> Jour. Amer. Med. Assn., 1905, xlv, 1156.

as well as the surgical tuberculous lesions, to bovine infection, but he nevertheless emphasizes their importance as immunizing factors against renewed infection with human bacilli.

Experience, experimental as well as clinical, among animals has also not revealed any hereditary transmission of specific "predisposition" to the disease, despite the fact that clinical medical treatises keep on speaking of "predisposition" which is transmitted from generation to generation. Speaking of specific predisposition, Baldwin<sup>1</sup> says: "Here again the bovine race gives a negative to the assertion that tuberculous infection necessarily involves a transmitted weakness or susceptibility. On the contrary, breeding from tuberculin-reacting cows is actually practised as of eugenic value in preserving the best stocks. The well-known Bang system has been on trial long enough in Denmark to have demonstrated its value, and is, I believe, the approved method of procedure in valuable dairies where tuberculosis is a serious menace." Harlow Brooks has shown that the progeny of tuberculous cows show no predisposition to the disease, as was already mentioned.

We know that all consumptives swallow tubercle bacilli, yet tuberculosis of the gastro-intestinal tract is not as frequent as opportunities for infection would lead us to expect. When infection of these organs does take place, the lesions remain local without extending to the regional lymphatic glands, as is the rule with primary intestinal tuberculosis.

Secondary tuberculosis of the skin is exceedingly rare in consumptives, although sputum reeking with tubercle bacilli is very often carelessly handled by them; and when it does occur, it runs a much milder course than lupus—primary tuberculosis of the skin. The well-known "pathologist's wart" and "butcher's wart" which, although of a tuberculous character, are of no significance, apparently because of old and dormant tuberculous lesions in some other parts of the body which confer immunity.

**Tuberculosis on "Virgin Soil" in Human Beings.**—While direct experiments on human beings are not available for obvious reasons, still some clinical facts are known which confirm the view just expressed. Bearing in mind that newborn infants are free from tuberculosis, no matter from what stock they are descended, we should expect that if tubercle bacilli were inoculated into infants, the resulting disease would run an acute and progressive course, just as is the case with experimental tuberculosis in guinea-pigs or rabbits. This is actually the case when during ritual circumcision among Jews the wound is infected with sputum from a tuberculous operator (Mohel.) The infant promptly becomes sick with tuberculosis and the disease runs an acute, rapid and fatal course, the regional lymphatic glands being implicated. This is a drastic contrast to the mildness of the "patholo-

<sup>1</sup> Amer. Jour. Med. Sci., 1915, cxlix, 882.

gist's wart" in the adult, which is also acquired by inoculation of tubercle bacilli into a wound. Woods Hutchinson<sup>1</sup> says that the first thing that struck him on visiting American Indian children's schools and reservations was the large number of individuals, both adults and children, showing huge scars in the neck or enlarged glands; next, he found a strong tendency among Indian children to acquire tuberculosis of an exceedingly rapid and fatal type.

On the other hand, Baumgarten injected cancerous adults, who may be assumed to have been infected with tuberculosis during childhood, with virulent bovine tubercle bacilli, and Klemperer injected similar microorganisms into tuberculous persons, without any deleterious results (see p. 50). These authors sought to prove that bovine tubercle bacilli are harmless to man, but in truth they confirmed experimentally that infected individuals are immune to superinfection. In infants, tuberculosis, when it causes disease, appears as a general disease similar to typhoid or septicemia; as a metastatic infection with deposits of tubercles in various parts of the body, like pyemia; or as an acute pneumonic or bronchopneumonic process, fatal in the vast majority of cases. The explanation for this phenomenon is that in the infant there occurs a primary massive infection of an organism that has been free heretofore from the tuberculous virus—real virgin soil. The same is true of primitive peoples who have never been infected with tubercle bacilli—when they are infected as adults, the disease pursues an acute and fatal course almost invariably.

**Phthisis as a Manifestation of Immunity.**—From the experimental and clinical data arrayed here, it is clear that neither infection with tubercle bacilli nor predisposition is alone capable of producing phthisis. To each one who has become phthisical, there are many who have been infected with tubercle bacilli and remained healthy in the clinical sense. In fact, spontaneous infection acquired during childhood appears to render the body immune against further and renewed exogenic infection with the same bacilli.

It is also clear that phthisis occurs only in individuals who have been infected with tuberculosis during childhood, but have remained healthy till adolescence. In other words, phthisis occurs only in persons who have been immunized by an earlier infection. In fact, it is in itself a manifestation of immunity, otherwise the patient would succumb to acute general miliary tuberculosis, as do those who have not been immunized by earlier mild infection. This immunity is apparently sufficient to protect the individual under ordinary circumstances, but under certain conditions it may fail, and the person may be re-infected either from without, the tubercle bacilli being so ubiquitous that we can hardly escape them; or from within, by the proliferation of the bacilli that have been harbored in "healed" or quiescent foci, through metastasis.

<sup>1</sup> New York Med. Jour., 1907, lxxvi, 624.



**Failure of Immunity.**—Acquired immunity in contagious diseases is hardly ever absolute—it is only relative, sufficient for the ordinary conditions of life and failing during emergencies. The same appears to be true of the immunity acquired during childhood by infection with tubercle bacilli. It protects the average person against exogenic reinfection with tubercle bacilli, and moderate failure of immunity permitting reinfection does not result in general tuberculosis, but only in phthisis—the most vulnerable organ in the body succumbs, while the others are still more or less protected.

There seems to be good evidence to the effect that the outcome of the infection which practically everybody passes through during childhood depends, in a large measure, on the extent of the microbial invasion. When the dose is small, immunity is the result, immaterial whether the initial lesion has healed completely, the bacilli being destroyed and the lesion cicatrized, or not. When there remain calcareous foci containing virulent tubercle bacilli, they remain innocuous as regards their host, and are probably an even better foundation for immunity. But when the initial bacterial invasion is massive it may cause hematogenic tuberculosis of the glands, bones, or joints during childhood; or when the resistance is very low, fatal tuberculosis of any organ, especially the lungs, meninges, etc., may result. But even massive infection may be kept in check till adolescence when, under certain exciting causes, the lesion flares up again and phthisis is the result.

**Immunity through Bovine Infection.**—Some authors have been inclined to attribute the immunity observed in most adults to infection during childhood with the bovine type of bacilli which protects the individual against superinfection with bacilli of the human type. Clive Riviere<sup>1</sup> even advocates the immunization of humanity along these lines. He says that “until human sources of infection can be practically eliminated, or artificial immunization becomes an accomplished fact, infection with the bovine bacillus through the use of a well-mixed milk remains our best ally in the campaign against tuberculosis.” We have seen already that bovine infection is fatal only on exceedingly rare occasions. That it may protect against infection with the human type of bacillus is made highly probable by the rarity of phthisis in surgical tuberculosis. “Very significant in this respect also are the figures of McNeil for Edinburgh where, as shown by Fraser and Philip Mitchell, tuberculosis of bovine origin is particularly rife. Comparing Edinburgh with Vienna he finds the incidence of tubercle infection higher in the former for children up to the age of four years, and this in itself is highly suggestive of milk infection; but the valuable comment on this is the fact that the mortality from phthisis in Vienna is nearly three times as high as that for Edinburgh. Indeed, the high incidence of abdominal tuberculosis,

<sup>1</sup> Brit. Jour. of Tuberc., 1914, viii, 83.

and the low mortality from phthisis, is characteristic of Great Britain as a whole when compared with other civilized countries of Europe, and this may well bear the interpretation that it is the early bovine infection which protects against the inroads of pulmonary tuberculosis caused by the human strain of tubercle bacillus."

**Nature of Predisposition to Phthisis.**—Obviously, the evolution of phthisis does not depend alone on the intensity of the infection during childhood. The character of the soil invaded by the bacilli is perhaps more important. Some succumb to hematogenic tuberculosis even as a result of a mild infection, harmless to the average individual, which indicates that predisposition was a stronger factor. In what this predisposition consists we are in the dark, though some factors are known to reduce the natural resisting forces to a minimum. Thus, as we have already shown, certain occupations, especially those involving the inhalation of dust, prepare the soil for the proliferation of the bacilli by reducing the vitality of the lung locally. Perhaps shortening of the first rib and ossification of the first costal cartilage are instrumental in this direction in some persons.

Failure of immunity may be due to various complex biochemical changes in the body with which we are unacquainted at the present state of our knowledge. This is seen in children who have been infected but who thrive in spite of it, until an attack of measles, whooping-cough, etc., which is accompanied by a failure in allergy, as is evident from the negative outcome of the cutaneous tuberculin test during the active stage of the disease, flares up the latent tuberculous focus and tuberculous bronchopneumonia results. Other febrile diseases may act in the same manner, but we do not as yet know the exact nature and effects of these biochemical changes in the body following contagious disease.

The nature of predisposition is the stumbling-block of the theories of phthisiogenesis. Clinical, demographic, and experimental observations have not cleared up these important problems. It appears that no single predisposing factor, nor a combination of several factors, will fit most cases. As has been pointed out by Martius,<sup>1</sup> the predisposition of the individual is, after all, not a specific entity, which is possessed by those who are attacked by phthisis, and lacks in those who escape the disease despite infection. It appears to be a complex affair: In each individual case there are a number of anatomical and physiological factors which may each alone, or several in combination, decide under certain conditions whether the person is to become phthisical, and even these factors are subject to great oscillations, and may combine differently under different conditions. From this point of view everybody is predisposed to tuberculosis, but there are many important differences in the resisting powers of different individual persons which depend on the number, intensity and accidental com-

<sup>1</sup> In Brauer, Schröder and Blumenfeld's *Handbuch d. Tuberkulose*, i, 395.

binations of the various predisposing factors which, by themselves, are influenced by certain vital, biological oscillations occurring during the lifetime of the individual. We thus have gradations of predisposition from the strongest degree of vulnerability to the highest degree of immunity.

**Endogenic and Exogenic Reinfection.**—Considering phthisis as a disease which develops only in an organism which has been immunized by an earlier infection which has left a latent or “healed” tuberculous focus in some part of the body, the problem arises whether the flaring up of the local lesion in the lung is caused by a new infection from without by the invasion of new bacilli, or from within by metastatic migration of bacilli which have been kept dormant for years until the immunity they conferred fails for some reason.

Experimental findings on this point are somewhat conflicting. Orth and Rabinowitsch<sup>1</sup> have found that when guinea-pigs are mildly infected with small doses of mildly virulent tubercle bacilli which cause only local tuberculous changes, the effect produced is that a second infection with virulent human bacilli does not cause the usual generalized tuberculosis, but pulmonary tuberculosis results, bearing some analogy to pulmonary tuberculosis in human beings. In rabbits, which react to human bacilli in a manner similar to that of man, more than guinea-pigs, they produced in this manner chronic tuberculous lesions in the lungs. Hamburger, Bartel, Levy, and others have confirmed these findings. This would indicate that phthisis is due to exogenic superinfection.

That the outbreak of phthisis is due to an autogenic, or metastatic reinfection has been maintained by Behring, according to whom the primary infection takes place through the gastro-intestinal tract during childhood, the bacilli remaining latent till stirred into activity by some exciting cause. But if this was the case we should expect that pulmonary tuberculosis due to bovine bacilli would be very frequent, considering that at least 10 per cent. of infections during childhood are caused by this type of microorganism. As it is, there have been reported very few cases of phthisis in which the bovine bacillus was found exclusively. It has been suggested that those infected with bovine bacilli are immune against human bacilli, and they are the ones who escape phthisis despite tuberculous infection, but this would have to be proved.

Römer and Much maintain that their investigations lead them to the conclusion that reinfection is always endogenic, or metastatic from existing tuberculous foci within the body. “We know” says Much,<sup>2</sup> “that a tuberculous organism is not susceptible to, in fact it is immune against, superinfection from without. We must also admit that when an organism is infected during childhood it passes through a precarious crisis, but it may survive this first infection and remain endowed with

<sup>1</sup> Drei Vorträge über Tuberkulose, Berlin, 1913.

<sup>2</sup> In Brauer, Schröder and Blumenfeld's Handbuch d. Tuberkulose, i, 247.

immunity. But during adolescence, when great demands are made upon the vital forces, the body may be overwhelmed by the bacilli and the most vulnerable organ in the body—the lung—succumbs; thus phthisis results. One who hesitates in accepting these ideas of reinfection from within should only compare phthisis with syphilis.”

There are analogous conditions known in pathology showing that an organism may harbor virulent bacilli without any harm to itself. Thus, the “carriers” of typhoid, diphtheria, and other bacilli may go around for years without showing any symptoms of disease, although they are a constant danger to others. But Texas fever illustrates this point even better. Cattle which survive an attack remain with the living virus within their bodies, but are immune against new infections, so that they may remain in infected pastures without any danger to themselves. But should they suffer from any secondary derangement, they may, as a result, experience an acute exacerbation of the process owing to sudden proliferation of the virus which has been dormant for a long time within their bodies.

There are similar clinical phenomena in man. It is known that infection with the malarial parasite protects against further infection with the same parasite from external sources, and for this reason the adult indigenous individuals in malarial districts are immune to malaria, as was shown by Koch. In some cases there occurs further infection in later years, and the result is a cachexia, a sort of malarial phthisis. But in such cases the initial infection must have been an especially strong and severe one. In syphilis this is even illustrated to a better advantage. Superinfections are very difficult, usually impossible; the integuments and mucous membranes cease to react to the syphilitic virus introduced from without while they are susceptible to their action from within. John A. Fordyce,<sup>1</sup> in a recent review of this subject, cites several other examples: “Levanditi has demonstrated that animals suffering with spirillary infection are immune to a new inoculation. Their serum has a high antibody content, but the blood still harbors parasites and is capable of producing a fresh infection in healthy animals. So with the serum of guinea-pigs inoculated with Nagana or Surra trypanosomes. This is trypanocidal for these organisms *in vitro*, but *in vivo* they have acquired an insensibility to the trypanolytic antibodies, for the blood and tissue of the animals still contain parasites. The same is true of human subjects suffering from sleeping sickness in whose serum trypanolytic, agglutinating and other protective bodies have been demonstrated. Carrying the analogy to syphilis we find that an individual may harbor spirochetes for forty or fifty years, while his skin and mucous membranes exhibit an insusceptibility to reinoculation under natural exposure. However, as soon as he is freed from his infection he is again in as susceptible a state as he was prior to his first attack.”

<sup>1</sup> Amer. Jour. Med. Sci., 1915, cxlix, 761.

We have shown that healed tuberculous lesions contain living and virulent tubercle bacilli; in fact even calcified foci contain them. It has even been questioned whether once infected with tubercle bacilli, the virus is ever absent from the body. And for this reason we may look upon phthisis as produced by endogenic reinfection. Thus, according to Römer, phthisis is an acute or subacute exacerbation of a latent or quiescent lesion in the lungs acquired by massive infection during childhood, the bacilli remaining dormant for years, but when the immunity which they conferred failed owing to some intercurrent disease, the lesion in the lungs flared up. That the specific immunity is not altogether lacking even under these circumstances is evident from the fact that the lesion remains localized for a long time in the most vulnerable of organs—the lungs. Phthisis is thus proof of immunity against tuberculosis. General miliary tuberculosis cannot develop in an individual who has been immunized by a previous infection with tubercle bacilli.

The question why adults are not immunized by mild infections as children are, has not been explained satisfactorily. We have already mentioned that adults hailing from countries where tuberculosis is unknown and where they could not have been infected during childhood because of the lack of tubercle bacilli, upon coming into cities and in contact with tubercle-laden surroundings—subjected to primary tuberculous infection—soon succumb to the acute forms of phthisis, like infants or guinea-pigs. Much attempted to explain it by saying that either the organism of the child alone is capable of evolving a sufficient quantity of immune bodies; or we must assume that an adult person coming from an environment free from tuberculosis to one which is tubercle-laden, freely going around among people among whom there are many bacilli carriers, is soon subjected to massive infection against which he does not possess sufficient powers of resistance. On the other hand, the sheltered child does not roam around among various people during the first years of its life and comes in contact with only a few bacilli, as long as there is no active case of tuberculosis at home. I may add that the suggestion made above to the effect that the immunization of humanity during childhood may be accomplished by the bovine type of bacillus, which is not as virulent as the human type, may be responsible for this salutary condition. But this problem has not yet been worked out.

**Summary.**—At the present state of our knowledge the following conclusions of Römer are justified. Tuberculous infections occurring during childhood, so long as they are not acute and fatal immediately, endow the organism with a heightened resistance against renewed infection with tubercle bacilli. The immunity thus produced is, as a rule, sufficient to protect the person against exogenic infection during later years. When, however, extraordinary physiological or pathological conditions permit the bacilli harbored within the body to proliferate because of the inadequacy of the failing immunity, which

becomes incapable of preventing metastatic reinfection, new tuberculous foci are formed, and again clinical phenomena of disease make their appearance. Experience shows that these metastatic reinfections mostly occur in individuals who were subjected to massive infections during childhood. The paralytic thorax is perhaps a local predisposing factor, although by itself it is in the vast majority of cases the result of a relatively strong infection with tubercle bacilli during childhood.

## CHAPTER VI.

### PATHOLOGY AND MORBID ANATOMY.

#### THE TUBERCLE.

TUBERCLE bacilli settling on susceptible soil offering suitable conditions for their growth, induce a specific proliferation of the fixed elements of connective tissue, capillary, endothelial, and probably also of the epithelial cells of the air vesicles. Acting as irritants and injuring the cells and the intercellular substances, they produce a productive inflammation resulting in the formation of a nodule, the specific granuloma termed tubercle by Laennec.

The tubercle is best studied in acute miliary tuberculosis, where it is encountered in its purest form. Throughout the lungs are scattered small, hard nodules. They may be gray and transparent, or yellowish-white and opaque. The transparent tubercles are smaller than millet seeds, while the opaque ones are as large or even larger. They are larger and more numerous in the upper parts of the lung where they grow better and more rapidly.

Microscopically, the tubercle presents a characteristic structure (Fig. 8). Primarily it is an avascular structure; with the growth of the cells, the bloodvessels and lymphatics in its neighborhood are compressed and obliterated. Its most peculiar characteristic is the large multicellular unit known as the giant cell. In thin sections, a fine network, the reticulum, is seen. The filaments are derived partly from extravasated fibrin, partly from curled fibrils of connective tissue, and partly from long, branching, interlacing processes of the cells, especially the giant cells, which have been described as looking like spider's feet, and also from newly formed connective-tissue fibrils.

Histologically, tubercles are classified as the epithelioid and the round-cell varieties, depending on the predominance of either of these two types of cells. The peripheral cells are arranged concentrically; near the centre they are larger, round, or oval, like epithelial nuclei. Scattered throughout are to be seen single lymphocytes with small, round nuclei and, in the typical tubercle, the polynuclear giant cell is located in the caseated centre. While the tubercle is often round, it may be of any form or shape and it usually sends out branches connecting it with the surrounding tissues. Most authors consider the round cells as lymphocytes which have wandered from the bloodvessels or lymphatics.

The so-called epithelioid cells arise through the proliferation of the connective tissue, and especially the endothelial cells in hematogenic

tuberculous follicles. They divide by karyokinesis and fission of the nuclei, and because the product is similar to epithelial elements, it is called epithelioid. Such follicles are mostly of microscopic size and consist mainly of this type of cell. They proliferate very slowly. With his theory of phagocytosis Metchnikoff, however, sees in these epithelioid cells derivatives of white-blood corpuscles, and inasmuch as they often show ameboid movements, they cannot be anything else than white-blood corpuscles. It has, however, been shown that these ameboid movements are no conclusive proof that they are of this origin.

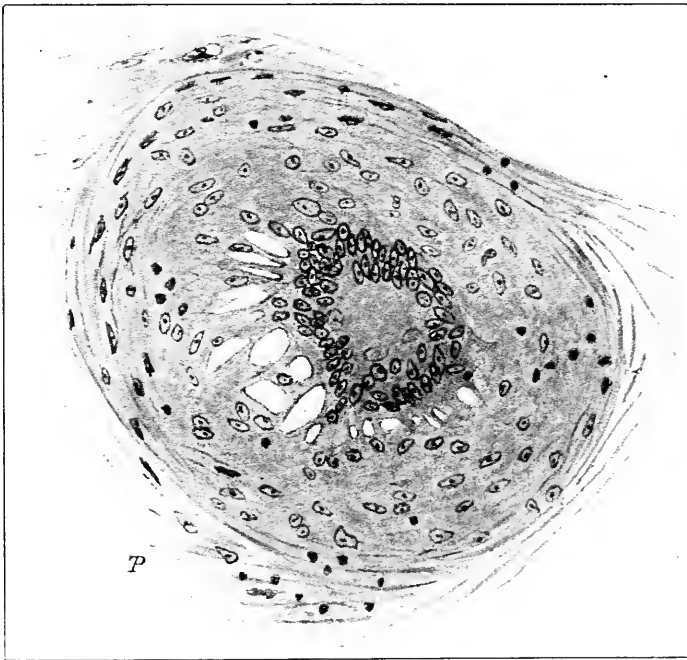


FIG. 8.—Microscopic tubercle. (Tendeloo.)

**The Giant Cell.**—The giant cell is polynuclear, with a stroma of fatty degenerated or even necrotic protoplasm. Its form and size are variable. It may contain as many as one hundred oval, spindle-shaped nuclei arranged concentrically like a crescent. The tubercle bacilli are mainly located in the giant cells (Fig. 8), where they may be seen singly or in clusters, usually at the inner side of the nuclei, or between the latter. They are, however, lacking in the centre of the protoplasm of mature giant cells; probably the process of necrobiosis affects the bacilli as well as the body of the cell.

The origin of the giant cells has been a debated subject. Some, like Weigert and Baumgarten, state that they are the results of karyo-



kinetic changes of the nuclei which retain their capacity for division while the protoplasm, owing to the necrobiotic effect of the tubercle bacilli, does not divide into separate cells. In fact, it is quite common to find in tuberculous foci cells with degenerated protoplasm, while the nuclei show an increased chromatin content. From this point of view the giant cell is a degenerative phenomenon. On the other hand, Metchnikoff sees in the giant cells one of the manifestations of phagocytosis: They are macrophages, or large active phagocytes, produced by the fusion of many epithelial cells with the object of fighting the invading enemy, the tubercle bacilli, with united forces. The part of the giant cell which has no nuclei is usually dead because of the noxious effects of the tubercle bacilli.

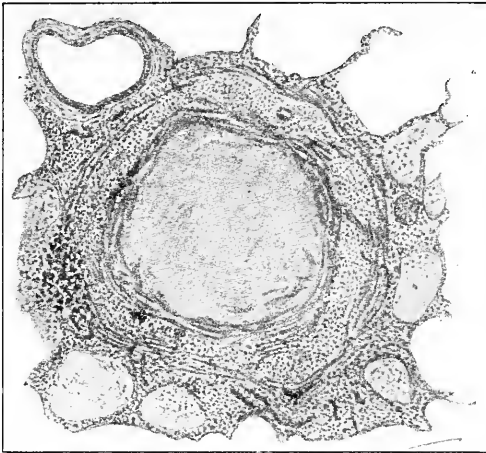


FIG. 9.—Cross-section of tuberculous bronchus. The lumen of the bronchus is completely filled with muddy but quite homogeneous caseous matter and the mucous membrane has completely vanished. The rest of the bronchial wall is very rich in cells and thickened. The thickening extends far into the neighboring alveoli. (Ribbert.)

Tubercle bacilli are mainly found in the giant cells, as we have already mentioned, and also in the epithelioid cells, while in the intercellular substance they are only rarely noted. In the caseous parts of the tubercle the bacilli are found at the periphery, while they are never seen in the centre. In the caseated giant cells they are found only in the parts which have retained their staining property.

**Caseation.**—The tuberculous follicles are avascular neoformations and their vitality is not durable. No new bloodvessels are formed in them, as is the case with most other new growths. They are usually located in the alveolar framework whence they compress the neighboring alveoli and finally obliterate them, and partly in the smallest lymph vessels, *i. e.*, along the walls of the smallest arterioles and bronchioles. In the arterioles a tuberculous obliterative endarteritis is formed and this alone, or in conjunction with thrombotic phenomena,

leads to occlusion of the vessel. In the small bronchioles caseous bronchitis may result, which may, however, arise primarily and lead to peribronchial tuberculosis secondarily. The bronchi become permanently plugged by their own secretions and by the irritative proliferation of their epithelium. The tuberculous growth compresses and destroys the elastic fibers, so that in the centre of the nodule there are only fragments of these tissues and often not even that, and air is completely excluded.

The necrotic tissue is thus converted into a whitish or muddy, yellowish-opaque mass; dry, often fragile, at times soft or even viscous in consistency. It has the appearance of dry or soft cheese.

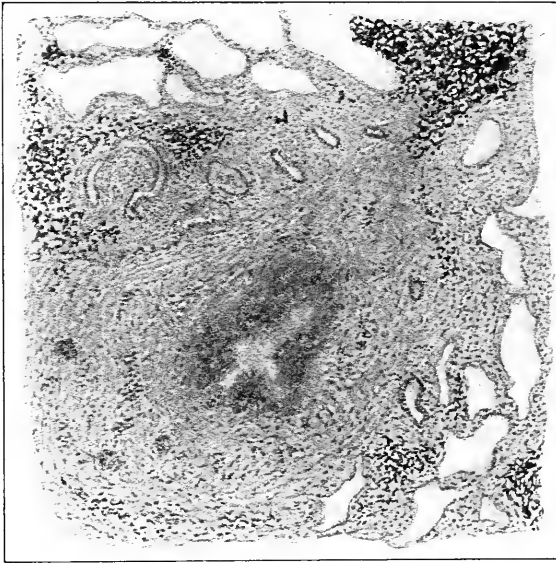


FIG. 10.—Indurated nodule in pulmonary tuberculosis. The solid nodule has a dark, caseous centre with irregular lacunæ. It consists of coarse connective-tissue fibers in which carbon particles are deposited in some places. A giant cell is seen in the middle and to the right, three others are seen to the left. (Ribbert.)

Microscopically, the cells are found to have undergone coagulation necrosis or fatty degeneration and are converted into a structureless mass of detritus which refuses to stain. At times, we make out between the remnants of the cells a filament, consisting of a fine network of granular fibrin, or true hyaline fibrin, the so-called "fibrinoid." Finally, a stage is reached when the débris of cells and fibrin become a homogeneous mass in which no structure is seen at all. This is true caseous matter.

Some have suggested that tuberculous toxins are specifically effective in causing necrobiosis of the affected cells, but this has not been proved. It must be emphasized that desquamation of epithelial

cells, necrosis, and caseation are not specific tuberculous changes. They are found also in various degrees of intensity in several other inflammatory processes in the lungs. Necrosis, especially coagulation necrosis, is also found in diphtheritic inflammation of mucous membrane, and caseation in gummatous changes. The caseous gummatous nodule can hardly be differentiated from the tuberculous.

**Calcification.**—The caseous matter may become surrounded by a layer of connective tissue—encapsulated—and then, by the exclusion of water, it becomes inspissated and much reduced in size. In time small granules of calcium are deposited until it becomes altogether calcified. Small calcified granules may coalesce into larger concretions until finally they are converted into a dry, solid, jagged, or fragile concretion which looks very much like chalk. These concretions often contain virulent bacilli. In general, it can be stated that it is never dissolved or absorbed by autolysis as is the case of other dead matter in the tissues. But caseous matter may be gradually permeated by fibrinous tissue and finally converted into a solid fibrous scar.

**Softening.**—Very often the tubercle, instead of calcifying or undergoing fibrosis, softens as a result of the action of proteolytic enzymes with which we are yet unacquainted. In this case there develops a puriform, thin liquid, without any pus cells but containing bits of cheesy matter, which is known as puriform liquefaction and “tuberculous pus.” In other cases real pus is formed, or a mixture of both liquids which is also known as tuberculous pus.

**Sclerosis.**—But the tubercle is not always destined to necrosis, caseous degeneration, calcification or liquefaction. In most cases in which phthisis does not develop at all, or is checked in its progress and healing finally results, the cells of the tuberculous nodule are converted into fibrous scar tissue through the agency of the proliferating connective-tissue cells. These connective-tissue cells are derived from two sources: From the cells in the neighborhood of the tubercle and from the tubercle itself. While making autopsies on persons who died from any cause pathologists have found that a large proportion have scars in their lungs and pleura, thus showing that an enormous number of persons have had tuberculosis which healed spontaneously. These healed or dormant lesions are responsible for the large number of persons obviously non-tuberculous, yet responding to the tuberculin test.

The fate of the tubercle depends on the intensity of the two processes of connective-tissue proliferation or sclerosis, and of caseation. In fact, the clinical course of the disease is mainly influenced by their relative intensity, the former being reparative and the latter destructive. If the exudative process predominates and progresses with rapidity, the tuberculous focus increases in size and clinical significance; but when the proliferative process predominates, the inflammation proceeds slowly and may even terminate in a cure through sclerosis. In chronic phthisis the two processes usually go hand-

in-hand; the reparative, manifesting itself by the proliferation of connective-tissue cells, is seen at the periphery of the tubercle, while the centre caseates. Pathologists then speak of fibrocaseous phthisis. In conglomerate tubercles the central foci may caseate while those at the periphery are healing by sclerosis and thus surround the lesion and prevent its progress by encapsulation of the cheesy centre which finally calcifies, as was already shown.

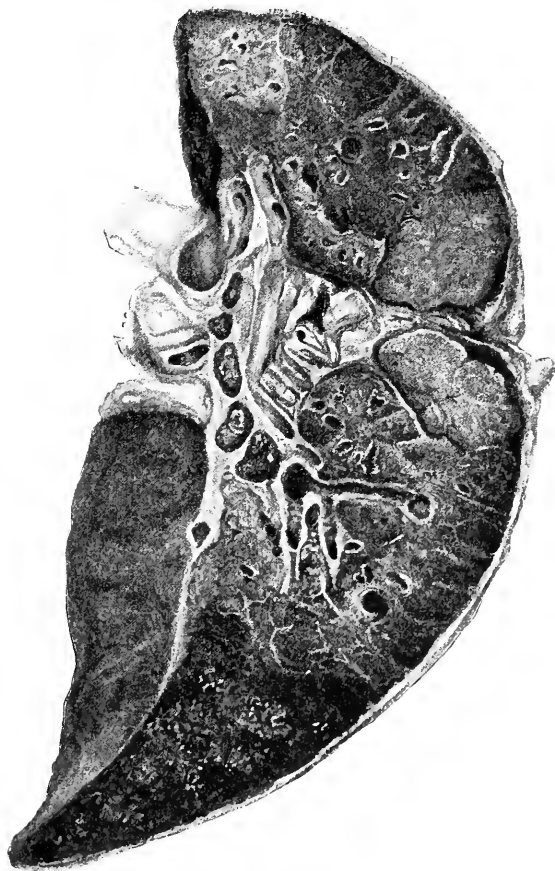


FIG. 11.—Caseous consolidation above. Red hepatization below. (Kast and Rumpfer.)

**Tubercles in the Lung.**—The first foci usually take root in the neighborhood of the apices and may remain there exclusively for a long time; in progressive cases, they may extend by the production of new nodules. They usually consist in a combination of both the productive inflammation in the form of nodular formation, and a pneumonic process. The first tubercles are found as single and isolated

nodules or groups around the bronchi and the bronchioles, and at times also around the walls of the larger bronchi and the bloodvessels—peribronchial and perivascular tubercles. According to the intensity of the affection and the resistance of the individual, the nodules enlarge and extend slowly or rapidly and new ones appear around them. Large conglomerations of tubercles may thus be formed. In progressive cases the tubercles do not remain separated for a long time, but by fusion of many the focus enlarges and extends. The central nodules sooner or later begin to disintegrate and are converted into caseous matter. But in most cases a sclerotic process may be detected which limits its progress, excepting in the very acute types of the disease.

The gross appearance of the typical tuberculous lesion in the lung presents a very variegated picture. In fact there are hardly two cases which look alike. The scar tissue surrounding the cheesy centres, or insinuating itself within many caseous and softened areas, is a very strong substance made up of thick fibers and can be recognized by its color. It is dark because particles of carbon derived from the inspired air are deposited in it and they cannot be expelled by expectoration because of their inability to reach the bronchial glands owing to the fact that the lymph channels are occluded or obliterated. It is therefore more or less dark gray or even black in color, which contrasts distinctly from the various other colors of the lungs. The distribution of scar tissue is variable. In some cases it is mainly in the centre of a group of tubercles, or it surrounds the caseated masses with extensive processes. A black, round or radiating scar may enclose a nodule the size of a pea or even larger, or several nodules. The cheesy matter is dry, and when old, calcified. This is very often found at the apex of clinically healed pulmonary tuberculosis.

Later the caseous matter softens and, when the degenerative process extends, reaching and implicating the bronchial mucous membrane, the softened débris may break through the alveoli or the bronchi. But in most cases sclerosis prevents the spread of the lesion and even encapsulates it with a more or less dense fibrous shell. Within the capsule the caseous matter dries up and finally calcifies, and it is stated that small foci may even be absorbed, though this is doubtful.

There has been quite some discussion as to the origin of ulcerations on the surface of the bronchial mucous membrane and in the parenchyma of the lung. Some have considered these as the points at which the infecting bacilli have entered with the inspired air and set up the disease; that these ulcerations represent the primary tuberculous lesion. As far back as 1876 Parrot pointed out that in all cases of tracheobronchial adenitis such a primary lesion may be found in the lung if carefully searched for. This is known among French pathologists as *la loi de Parrot*, Parrot's law. G. Küss<sup>1</sup> has confirmed Parrot's findings on extensive autopsy material, and more recently

<sup>1</sup> De hérédité parasitaire de la tuberculose humaine, Paris, 1898.

Anton Ghon<sup>1</sup> has found the same condition while making numerous autopsies. French authors refer to these primary lesions as *chancres tuberculeux*, and the enlarged regional glands which are almost invariably found, as *bubons d'emblée*.

Others maintain that there are many cases of tracheobronchial adenopathy in which such a primary lesion in the bronchioles or pulmonary parenchyma cannot be discovered at the autopsy. It is also shown that even when found it should not be concluded in all cases that this ulceration represents the point of entry of the bacilli. They may be due to extension of the peribronchial nodules which, when enlarging, have reached the mucous membrane, caseated it and produced ulceration. As was already stated in Chapter V, the problem whether phthisis is of hematogenic or bronchogenic origin rotates around this point to a large extent. The experiments of Bacmeister have shown conclusively that such lesions may be produced by the hematogenic route and that the primary lesion is not commonly in the mucous membrane. But this does not exclude infection of the mucous membrane. We have already shown that the bacilli may be deposited on the bronchial mucous membrane and pass through the lymph channels into the subepithelial tissue where they take root, without producing a lesion at the point of entry.

**Caseous Pneumonia.**—The nodular formations are not the only changes wrought by the tubercle bacilli in the lungs. There are also seen larger primary infiltrations which are pneumonic in character; in fact these distinguish phthisis from pure tuberculosis. These areas are of variable size, from that of a pea to that of an egg, or even larger. They are round, oval, leaf-shaped or lobular in arrangement (Fig. 12); they may be single or several may be found clustered together. They are pale, grayish and later muddy in color; at times they look like cheese. They are found in rapidly progressing fibrinous exudations which caseate quickly—caseous pneumonia. Real lobar caseous pneumonia is exceedingly rare. The diseased parts are voluminous, airless, heavy, like in the hepatization of lobar pneumonia.

Microscopically, there is found an albuminous mass in which fibrin, red-blood corpuscles and alveolar epithelium may be discovered, but the alveolar structure may still be made out at an early stage. When seen in the early stage we can follow the rapidly ensuing process of coagulation necrosis in the alveolar septæ. Tubercle bacilli are found in large numbers, especially at the periphery of the cheesy focus. The final result is always expulsion of the caseated and degenerated débris, leaving excavations, which will be discussed later on, excepting when the process involves but a very small area, and some authors say that a cure is then possible by absorption of the caseous matter.

Caseous pneumonia cannot always be differentiated from nodular tuberculous lesions, because when the nodules extend rapidly, as they

<sup>1</sup> Der primäre Lungenherd bei der Tuberkulose der Kinder, Berlin, 1912.

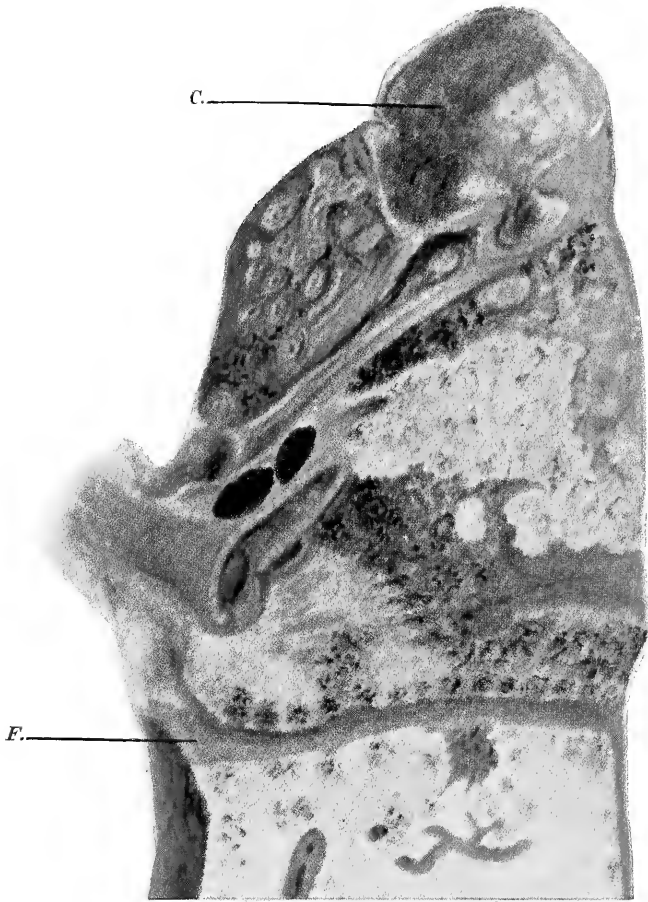


FIG. 1

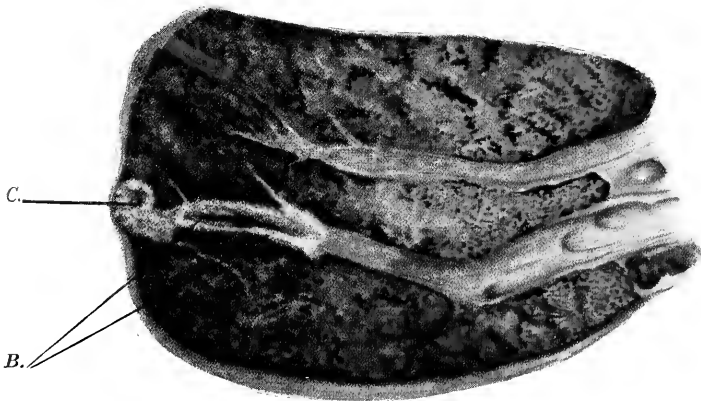


FIG. 2

FIG. 1.—*C*, cavity in the pulmonary apex. *F*, interlobar fissure. To the left of the cavity are seen peribronchial nodules. Lower parts are extensively caseated.

FIG. 2.—*C*, small caseous focus in the upper part of the apex. *B*, bronchus with caseated wall. The rest of the parenchyma is of normal air content, but anthracotic and showing black pigmentation. (Albert Fraenkel.)





do in some acute cases, they consist mainly of a conglomerate group of alveoli filled with exudate; the more rapidly the process progresses, the more they are coalescing and the greater the similarity to caseous pneumonia.

Beitzke<sup>1</sup> points out the main differences between tubercle and caseous pneumonia as follows: Caseous pneumonia is an exudative inflammation, while tubercle is a productive one. In the former there are therefore found loose exudate cells and fibrin, while in the latter solid tissue is found, and fibrin is almost never encountered. The exudate in caseous pneumonia lies in the lumen of the alveoli, while the tubercle is located in the interstitial tissues. In caseous pneumonia the elastic fibers remain intact, while the granulation tissue of the tubercle destroys them. These differences show the necessity for differentiation between the two processes. But etiologically they cannot be separated: Both are due to the same cause, both combine and affect the lung tissue, so that only the microscope can decide the intensity with which each is represented in a given lesion.

**Localization and Fate of the First Lesion in the Lung.**—The first lesion cannot be recognized at autopsies of cases on old chronic tuberculosis, and it cannot be definitely determined whether the disease has arisen by the hematogenic or aërogenic route, as has already been mentioned. It appears, however, that the initial lesion heals in the vast majority of cases. It may also happen that the initial lesion should be completely or partly healed in one lung, while the second lung becomes affected with progressive disease. The nodules undergo complete fibrous degeneration, become surrounded by connective tissue which often implicates the surrounding overlying pleura, a cicatrix is formed which contracts the part of the lung affected, resulting in those puckered scars so often seen at autopsies. Inasmuch as the lymph channels are obliterated, the pigment particles inhaled with the inspired air cannot be removed, and they remain in the connective tissue, thus causing slaty induration.

This mode of healing is not the rule. Often the focus caseates and is surrounded by a fibrous capsule; the caseous centre then softens, as has already been described.

**Extension of the Lesion.**—The morbid focus may erode a blood-vessel and thus break into the circulation, causing acute general miliary tuberculosis, but this is comparatively rare because of thrombosis of the supplying vessels. Usually the process extends by the invasion of the tissues in the immediate neighborhood of the initial tubercle. Even when some sclerosis takes place, or the old tubercles calcify, the extension may proceed unabated. Conglomerate tubercles, massive infiltrations which are complicated by pneumonic processes are thus evolved.

The bacilli spread along the lymph spaces and lymph channels

<sup>1</sup> In Aschoff's *Spez. pathol. Anatomie*, Berlin, 1913, ii, 299.

from the areas which have undergone pneumonic changes. This is proved by the fact that around old lesions there is often found a crop of new tubercles. In the same manner we explain fresh lesions in the neighborhood of old scars or calcified areas in the apex. Formerly it was thought that the latter are caused by new infections, or superinfections, but since we have learned about the immunity of the tuberculous to new exogenic tuberculous infections, we consider these as metastatic endogenic extensions of the process. These metastatic tubercles increase in number, coalesce, and finally caseate.

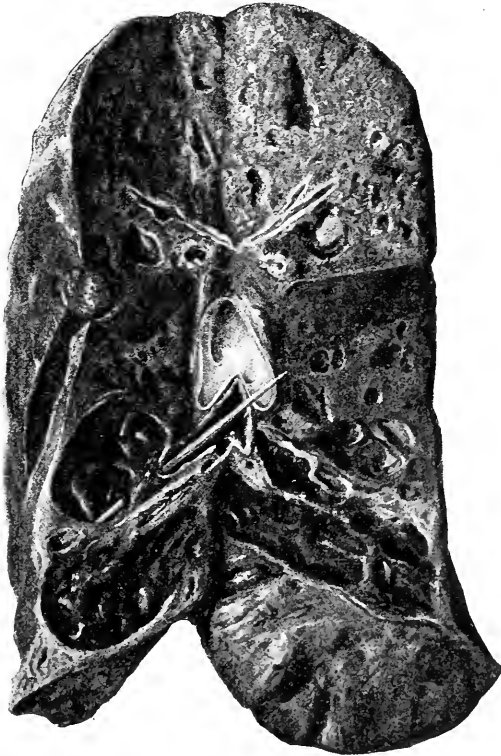


FIG. 12.—Caseous consolidation in the upper lobe and bronchiectasis in the lower lobe. (Kast and Rumpfer.)

At times the extension of the process proceeds along the peribronchial lymph channels and the result is a lobular arrangement of the focus, often looking like a mulberry. Some of these lesions, especially when exudation takes place, simulate the bronchopneumonic picture very much.

Metastatic extension of the process may also occur along the bronchial tubes and then it runs a rather acute and progressive course. When a necrosed focus reaches the inner surface of a larger bronchus and breaks through the mucous membrane, the caseated matter is

carried along the lumen of the tube and may be coughed out. But at times it is aspirated into the alveoli where it may produce a lesion similar to that of the primary infection. Inasmuch as in such cases we deal with larger numbers of bacilli, they may be distributed over larger areas. Most of these aspiration infections occur in the lower lobes of the lungs, but the metastatic infective matter may be carried to the apex by vigorous cough. These metastatic auto-infections may produce disseminated tuberculosis, but in the majority of cases a single area is infected and the lesion produced is of the caseous pneumonic variety, or indurated nodules result.

Dr. J. Kingston Fowler<sup>1</sup> has given in detail an account of the usual course of the secondary deposits in chronic or subacute phthisis as he found it while making numerous autopsies. He found that the first

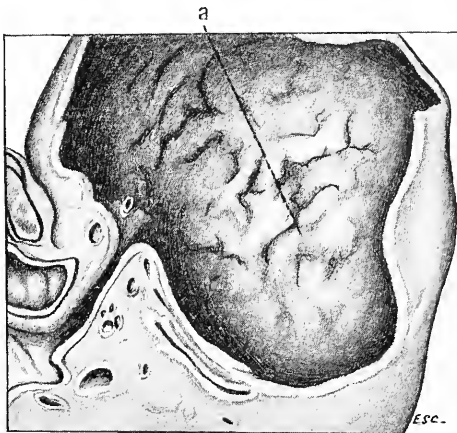


FIG. 13.—Tuberculous cavity (*a*) at apex of lung, showing its relation to a bronchus. (Adami and McCrae.)

deposit of tubercles is not at the extreme apex. It is most commonly situated from an inch to an inch and a half below the summit of the lung and rather nearer to the posterior and external borders, and spreads backward, this line of extension explaining the fact that the physical signs of tubercle are often first noticed over the suprascapular fossa. In front, the lesion corresponds to the supraclavicular fossa or to a spot just below the centre of the upper lobe, about three-quarters of an inch within its margin, and perhaps separated by an inch or more of healthy tissue. The second and less usual seat of the primary lesion is somewhat lower and more external, and corresponds to the first and second interspaces at the outer third of the clavicle. The lesion extends downward. The part which next shows tubercular deposit is the apex of the lower lobe (the middle right lobe being passed over), from an inch to an inch and a half below the upper and posterior

<sup>1</sup>The Localization of the Lesions of Phthisis, London, 1888.

extremity, and about the same distance from the posterior border, a spot nearly corresponding to the chest wall opposite the fifth dorsal spine, midway between the scapular border and the spinous processes. This lesion tends to spread backward toward the posterior border of the lung, and laterally along the interlobar septum. The extension in the lower lobe is almost always from above downward and by islands of deposit of racemose shape with healthy lung between. The second lung is seldom the seat of secondary deposits until the lower lobe of the first lung attacked is implicated. The lesions are usually situated in the same situations to those of the apex of the opposite side, but sometimes their site is close to the interlobar septum, midway between its upper and lower extremities, corresponding to the upper axillary fold. Extension in the lower lobe of the second lung follows the course of the lesions in the lower lobe of the first lung.

**Emphysema.**—The unaffected parts of the lung in chronic phthisis often show emphysematous changes; in fact, occasionally on removing the lungs from the thorax after death, they may be found so voluminous that the tuberculous lesion is not seen without a search. The surface of the emphysematous parts of the lung is usually puckered because of the traction exerted by fibrous bands and excavations within the organ; or, in localized emphysema, which is more frequent, the surface shows bullæ of various sizes.

This emphysema is compensatory. When one lung is extensively involved by the tuberculous process, the other undergoes vicarious enlargement, at times encroaching beyond the middle line; when both lungs are affected, the unaffected parts become emphysematous. It appears that this is strictly for the purpose of enlarging the alveolar surface of the parts which remain intact and thus increasing the breathing surface. In fact, microscopic examination of the emphysematous parts of the lung shows that there is no degenerative atrophy of the alveolar septæ and bloodvessels, as in true emphysema. The alveoli are simply distended.

**Cavitation.**—When the caseated and softened detritus, affected by certain chemical changes, becomes undermined in various directions, blocks of dense tissue are loosened and cast off, then expectorated, leaving vacant areas in the lungs which communicate with one or more bronchi. The walls may appear sinous, pouchy and covered with a caseous or purulent material and detritus of disintegrated tissues, or covered with a pyogenic membrane. In some cases they are smooth and glittering, all of which depends on their mode of origin.

The excavations in phthisis may be single or multiple and they are mostly located in the upper parts of the lungs, the apices. They may be the size of a hemp seed to that of a fist, and in rare cases the complete lung is excavated, leaving a thick shell of the pleura. William Ewart<sup>1</sup> pointed out that excavation is especially prone to attack

<sup>1</sup> Brit. Med. Jour., 1882.

definite regions of the lungs. The apex of the lower lobe is thus affected at a date anterior to the implication of the lower parts of the upper lobe. The base and anterior border of the lower lobe are least prone to excavation, just as these parts are altogether the last to be involved in the tuberculous process.

The question whether true bronchiectatic cavities may occur in phthisis has been debated. Ewart denied such a possibility, and when found, he considered it purely secondary to the undue strain thrown upon the spongy structures which escaped disease. But more recent investigations have shown that they may be found. Delafield and Prudden found them very frequently. The superficial layer of an affected bronchus may be cast off while the process of caseation goes on in the deeper layers. In fact, cavities may occur without the destruction of the inner bronchial lining. When the tuberculous process proceeds slowly and proliferation of tissue is more active than necrosis, the bronchi dilate cylindrically and, because the more resisting elements—cartilage, elastic fibers, and muscles—perish, only an unsupported, smooth or slightly ulcerated mucous membrane remains, which yields to the expiratory pressure of the air during cough. These excavations are usually cylindrical or round in shape. They may be considered true bronchiectatic cavities.

When multiple, the separating walls of cavities may be gradually destroyed and a sinous vomica is thus formed. The large vessels and the unaffected bronchi resist the destructive process for a long time and remain as cylindrical trabeculæ, traversing the cavity in various directions. These tough septæ and bridles are, however, not always remnants of persisting bronchi and bloodvessels. William Ewart has shown that they are more often chiefly composed of condensed airless lung, representing the remains of collapsed alveolar tissue originally separating discrete cavities. When finally these are also destroyed, only ridges and stumps of fibrous tissue remain within the cavity, and also septæ which separate accessory excavations communicating with the main cavity.

Only a small proportion of the cavities are bronchiectatic in origin; the vast majority arise through the caseated and hepatized pulmonary parenchyma and expulsion of the necrotic tissue by expectoration. They have irregular, ragged walls on which there are attached pieces of necrotic tissue of various dimensions, bands separating remnants of interlobular septæ of the lung. Within the cavity there are often found some large necrotic lumps of tissue or sequestra which are too large to be expelled through the communicating bronchus. On rare occasions a cavity is formed when a large part of caseated pulmonary parenchyma is sequestered *in toto*. In case the cavity is derived from a small caseous peribronchial or bronchopneumonic focus, it is small, more or less circumscribed and round. But when it is derived from a larger pneumonic process it is large from the start and irregularly limited. But small excavations may fuse, coalesce and form

large, pouchy cavities. The septæ which separate them fade away and a large, ragged excavation is formed; its walls are covered with a pyogenic membrane, consisting of granulation tissue and secreting tuberculous pus, like a chronic abscess:

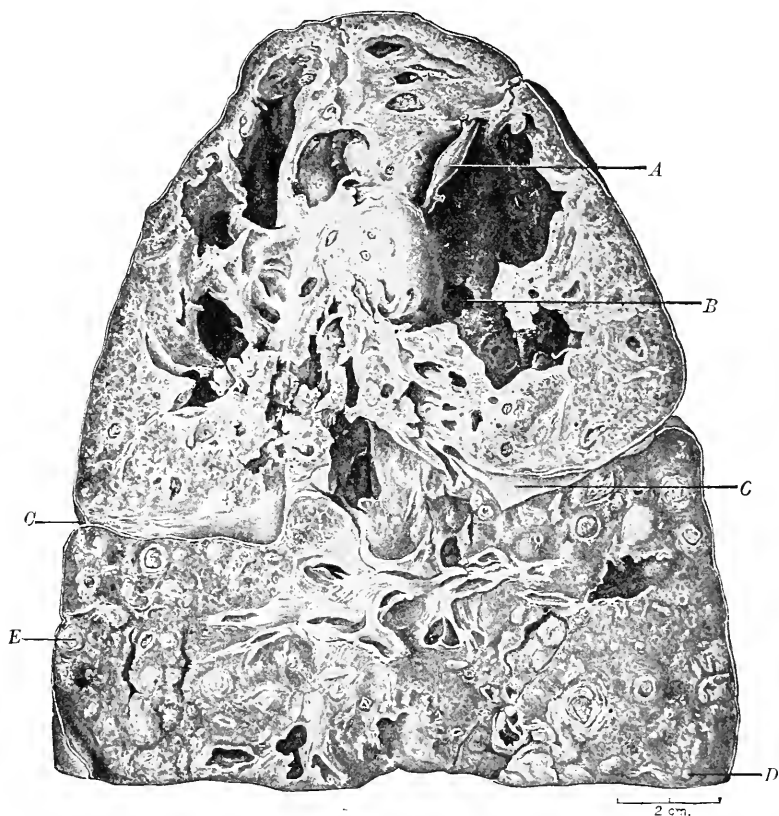


FIG. 14.—Left lung, superior lobe, and upper part of lower lobe, the former containing a number of communicating caverns, brought about by tuberculous infiltration, caseation, and evacuation of the contents through the bronchi: *A*, aneurismal dilatation of an artery spanning one margin of a large cavity; *B*, communication with another cavity; *C, C*, thickened and adherent pleura between the two involved lobes. The pleura over both lobes is thickened, and at the autopsy the cavity had been obliterated by universal adhesion; *D*, the pointer from the letter *D* leads to a small group of tubercles in which caseation is just beginning; *E*, a fused group of tubercles, farther advanced than at *D*. (Hare.)

William Ewart thus describes the walls of tuberculous cavities which have been freed from secretions and débris: Internally the surface is lined with a grayish false membrane, often of appreciable thickness, but in other cases possessing a little more substance than the bloom of a fresh fruit. In either case it is readily detached and exposes a layer which constitutes the inner and vascular portion of the

capsule, the outer portion of which is purely fibrous. The relative thickness of these three coats varies according to the age of the cavities and to the degree of irritation under which they may be placed. The chief features of tuberculous cavities are: (1) Absence of protecting epithelium; (2) gradual decay, leading to the formation of a necrotic layer (pseudomembrane); (3) gradual fibroid growth from without constituting the so-called capsule.

Formerly it was stated that cavitation implies mixed infection. T. Mitchell Prudden's<sup>1</sup> experimental investigations have shown that injections of pure cultures of tubercle bacilli into the trachea of guinea-pigs and rabbits produced pulmonary infiltrations; when streptococci were added, cavitation was produced. But more recent investigations tend to show that tubercle bacilli alone are capable of producing excavations. In this country Ira Ayer<sup>2</sup> found cavities in the lungs of rabbits after injecting intratracheally massive doses of a suspension of tubercle bacilli containing many coarse clumps. Bacmeister's experiments also showed that in animals in which tuberculous infection produces no cavitation, pressure on the apex will produce them (see p. 87), and that mixed infection is not necessary for the purpose. The pyogenic microorganisms found in the walls and secretions of tuberculous cavities are now explained as secondary implantations of these organisms after cavitation has taken place as a result of the action of the tubercle bacilli.

In slowly progressing or stationary cases a wall of connective tissue, even of non-tuberculous granulation tissue, may form around the excavation, and the necrotic parts within are cast off and expectorated, leaving a smooth cavity. On the other hand, in progressive cases, the necrotic process digs itself deeper and deeper into the parenchyma and the cavity keeps on enlarging and may attain extensive dimensions. With this process, non-tuberculous infection often takes place through the invasion of streptococci and staphylococci and other microorganisms which invade the walls. Here mixed infection is frequently very effective in extending the diseased area. The pleural layers over superficially located cavities are usually united by dense adhesions.

These cavities have a tendency to enlarge in the manner just described, but on rare occasions they may shrink because of vigorous sclerosis around the lesion which causes contraction. It is more common that the walls should remain smooth and quiescent for many years and, like a chronic abscess, discharge externally through a narrow sinus. But even caseous, ragged cavities may expel the necrotic tissue completely and permit the proliferation of connective tissue around the walls. Healing may thus result, the spongy condition of the adjacent lung favoring contraction. But such a course

<sup>1</sup> New York Med. Jour., 1894, lx, 1.

<sup>2</sup> Jour. Med. Research, 1914, xxv, 141.

is less likely to occur when the excavation is extensive, owing to the surrounding caseous pneumonic processes which usually show a tendency to progressive decay.

In extreme cases in which the excavations are extensive and the formation of connective tissue is vigorous, implicating the subpleural structures, the entire lung may be destroyed and reduced to the size of a man's fist. In these cases the diaphragm is pulled upward and with it some of the abdominal viscera, especially the liver and stomach. The mediastinum is pulled over to the affected side, pushed along by the unaffected emphysematous lung. Complete dextrocardia may be found in such cases, with the tuberculous lesion in the right lung; in left-sided lesions the heart is often pulled to the left and upward.

**Closed Cavities.**—Occasionally cavities are found in the pulmonary parenchyma which do not communicate directly with a bronchus, either because the lumen is occluded with the products of the exudate, or connective tissue has proliferated just at that point and plugged up the passage to the bronchus. Such a closed cavity may open up secondarily when the plug is removed from any cause. Perfectly closed cavities in the anatomical sense are not frequently seen, at any rate, not as frequently as clinicians make such a diagnosis.

**Hemoptysis.**—When the process of caseation and softening involves one of the bloodvessels, which very often traverse the walls of cavities, ulceration may extend to the vessel, causing profuse and fatal hemorrhage. The walls of the exposed vessel become thinner and thinner and finally erode. Because of the loss of support due to the progressive inflammatory decay of the surrounding pulmonary parenchyma, it finally yields to the intra-arterial blood-pressure.

More frequently hemorrhage occurs after the formation of an aneurismal dilation of some branches of the pulmonary artery traversing the walls of the cavity (Fig. 14). The diseased arterial wall yields to the pressure, gives in first without rupturing owing to the withdrawal of support of the exposed side, and a sacculated aneurism results; rarely a fusiform aneurism results from the uniform dilation of the artery. Douglas Powell points out that the fibrotic cavities of old standing are more likely to develop aneurism, and that aneurism is more especially found on the exposed side of vessels which are partly buried in indurated tissue. These aneurisms vary in size from that of a pinhead to that of a plum; usually they are single, but there may be more than one and, in rare cases, more than twenty have been found in one lung.

Because organized clots and thrombi obliterate the vessel, hemorrhage is comparatively rare. In small cavities the effused blood may by itself prevent further hemorrhage, provided the communicating bronchus is temporarily plugged, or is naturally of a narrow caliber. But most cavities are large and when a vessel ruptures, hemorrhage of great violence takes place.



**Rupture of a Cavity into the Pleura.**—When a rapidly progressing excavation is located superficially in the lung and reaches the surface, the pleura may caseate and rupture. In acute cases in which there is no time for the formation of adhesions between the pleural layers, a loss of continuity in the latter opens up a cavity and permits the escape of its contents as well as air into the pleural cavity. Pneumothorax is the result, and when this has lasted for some time, serous, and purulent effusions—hydropneumothorax, pyopneumothorax, etc., are formed. These are exceedingly rare in slowly progressing cases of phthisis because adhesive pleurisy results before rupture of an excavation takes place. In old cases I have observed that when pneumothorax does occur the rupture often takes place into the pleura of the side that was only recently implicated.

**Reparative Processes.**—We have already spoken of the process of repair that goes on hand-in-hand with the process of destruction in phthisis, and which is found to a certain degree in all cases excepting those of the most acute types. Judging by the large proportion of persons who at the autopsy are found with fibrous scars in the lungs and pleura, as well as with calcified foci in the parenchyma and glands, it becomes a convincing fact that more tuberculous lesions in the lungs are healed than progress to caseation and softening. It has also been found that many cases of these “healed” tubercles contain virulent tubercle bacilli and thus remain a constant source of danger: They may flare up at any time and again begin to activate, or by metastasis create new tuberculous foci in the adjacent or distant parts of the lungs or other organs.

Tendeloo<sup>1</sup> gives the following details about the reparative processes in pulmonary tuberculosis:

1. Every fibrous focus is to be considered as an old tuberculous lesion.

2. Calcification removes all danger of the further spread of the lesion. (This is not in agreement with the views expressed above and which are accepted by most authors.)

3. A fibrous capsule separates quite effectively its caseous contents from the rest of the parenchyma of the lung, and the process may remain quiescent for a long time. As long as there remains caseous matter within the capsule, or non-fibrous tuberculous tissue, there is always danger that the caseous focus may extend beyond the fibrous capsule, and also that the decay of the latter may favor a further extension of the tuberculous process by growth and metastasis. As long as the bacilli remain virulent in the lesion, and there are connections between the contents of the focus and the surrounding pulmonary parenchyma through lymph spaces, they can grow under certain circumstances and induce pathological changes in other parts of the lung. On the other hand, a fibrous capsule interferes with medication reaching the lesion.

<sup>1</sup> In Brauer, Schröder and Blumenfeld's *Handbuch der Tuberkulose*, 1915, i, 98.

4. A fibrous capsule has the same significance for an excavation. But in this case other dangers are added: As long as the cavities contain caseous matter, bronchogenic metastasis is threatening because there are always virulent bacilli in the caseous matter. The dangers of softening are greater in excavations communicating with the bronchi because the air has free access to their contents and may bring in other microorganisms, thus causing mixed infections.

5. Healing of a cavity is possible when it is cleared of its contents and the walls granulate. Small vomicæ may heal when their contents are evacuated and the walls shrink. In more extensive excavations there always remains some vacant space. When no open lesion remains, the elastic fibers and bacilli disappear from the sputum.

Ewart points out that whereas in other organs the obliteration of abnormal spaces is effected by free granulations arising from the bottom of the cavity, surface granulations are practically absent from tuberculous excavations. Still, he holds that, if freely drained, they may granulate successfully and the walls finally adhere. This is in agreement with the more recent views of Tendeloo. But this is more likely to be seen in small vomicæ, while in the large ones the air and fluid contents offer obstacles to perfect contact of the surfaces.

In general, we may consider the productive tissue changes as salutary, while the degenerative—caseation and softening—as phenomena lurking with dangers. Still, even in the latter healing is possible through calcification or the removal of the products of tissue disintegration from the air passages. It is doubtful whether caseous matter can be absorbed, though some insist that this is possible. Exudative tuberculosis may terminate favorably or unfavorably, according to its progress along the lines of absorption, or in other forms, caseation and softening, and elimination with the expectoration or by calcification.

It thus appears that even extensive tuberculosis may become quiescent, although we cannot speak of healing and *restitutio ad integrum* in the anatomical sense. It must always be borne in mind in this connection that the anatomical changes are not the only ones which decide the outcome of the disease in most cases.

**Pathological Changes in Other Organs.**—The glands, especially those in the thorax—the bronchial, tracheal and mediastinal—and of the mesentery are very often affected in children and adults who suffer from phthisis, more often than is generally appreciated. In fact, it may be stated that the tracheobronchial glands are affected in nearly every case of phthisis. On careful and painstaking search small, microscopic tuberculous foci are often found in apparently unaffected glands; but the majority are swollen, enlarged and many are softened while others are calcified. In children these tuberculous glands very often give no clinical indication of their implication; in fact, it is at times difficult to discover any changes in the bronchi and parenchyma on cursory examination at the autopsy. Still, these

glands are frequently a source of trouble, not only in causing symptoms of tracheobronchial adenopathy, but also because these conditions are to be considered the forerunners of phthisis in the adult, though some look upon them as possible immunizing agents against reinfection in later life.

By pressure these enlarged glands may cause stenosis of the main bronchus in children, while in adults it is less likely to occur because

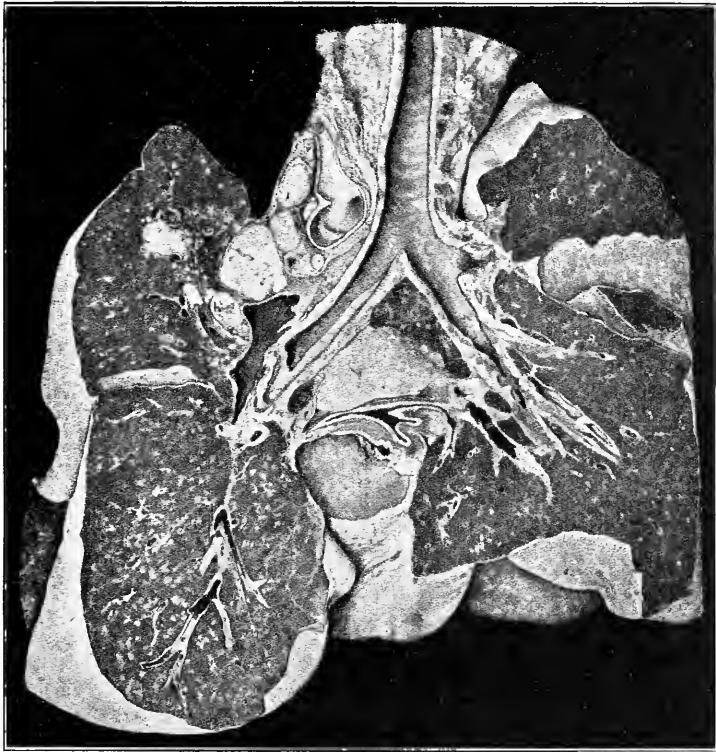


FIG. 15.—Primary caseous focus in the left upper lobe with miliary tubercles in its vicinity. Caseation of the regional lymph nodes of the left upper lobe. Caseation of the upper tracheobronchial lymph nodes. Acute miliary tubercles in the lower tracheobronchial lymph nodes. Over both lungs disseminated tubercles are to be seen. The upper tracheobronchial and bronchopulmonary lymph nodes in the right side are free from pathological changes. (Anton Ghon.)

the bronchi are firmer. But the smaller bronchi may be compressed in adults as well as in children. In the latter, suppurating glands at times perforate the trachea, bronchi, pericardium, or esophagus, causing sudden death, tuberculous bronchopneumonia, etc.

The mesenteric glands are only rarely affected in adults, even in those who have tuberculous ulcerations of the intestines, but in children they are often found to be the seat of tuberculous changes, particularly with bacilli of the bovine type.

**The Larynx.**—The larynx shows tuberculous changes in at least one-third of cases of phthisis. Proliferative and caseous, as well as ulcerative, lesions are found. These infections are usually secondary



FIG. 16.—Tuberculous pleural adhesion. At the lower part of the drawing is to be noted that the subcostal cellular tissue is very much reduced in quantity. Above it the new membrane is developed at the expense of the visceral pleura and shows a layer of tuberculous follicles. The fibrous tissue gradually extending upward and coming in contact with the lung without any sharp line of demarcation between them, is already old, well organized in parallel bundles and passed by numerous bloodvessels. (Chantemesse and Courcoux.)

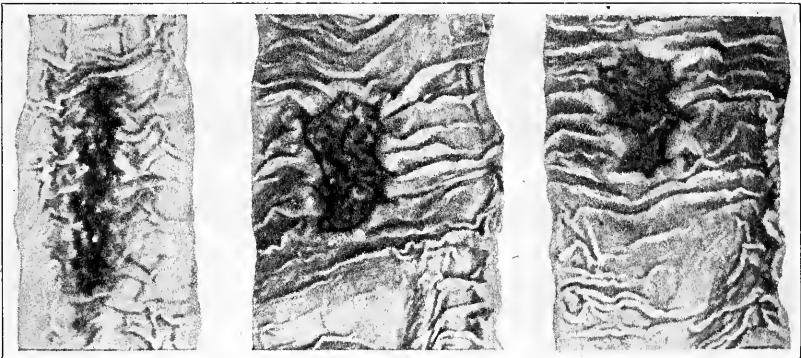
to tuberculosis in the lungs; primary tuberculosis of the larynx is exceedingly rare; in fact, some authors deny that it ever occurs. In many cases of laryngeal tuberculosis the trachea is also the seat of specific ulcerations.

**The Pleura.**—The pleura is implicated in nearly every case of phthisis. A large proportion of cases are preceded by pleurisy, moist or dry, but even then it is usually secondary to extension of some small lesion in the lung. Pleural adhesions are found at the autopsy in nearly all fatal cases of phthisis, excepting those running an exceedingly acute course. In some cases they are so dense and compact that it is difficult or impossible to remove the lungs without injuring the pleura. Sometimes the pleura is thickened all over; in many only partly, especially over the seat of the main lesions, and also at the base where thickening of the diaphragmatic pleura is not uncommon with resulting elevation of the diaphragm. Many fibrous bands are often seen extending from the pleura into the parenchyma of the lung. The adhesions may be lax and easily separated, but in many cases they are dense, and when extensive the thick pleura may surround the lung like a shell. On rare occasions the pleura is even

FIG. 17

FIG. 18

FIG. 19



FIGS. 17, 18, and 19.—Tuberculous ulcerations of the intestines. (Tendeloo.)

found calcified in places, or very extensively. Very frequently thickening of the pleura between the lobes of the lung is found. All these adhesions are great hindrances to the induction of artificial pneumothorax for therapeutic purposes. On the other hand, they prevent the occurrence of spontaneous pneumothorax through rupture of the visceral pleura over the site of superficially located pulmonary lesions, and when pneumothorax does occur, it is only localized. Sero-fibrinous pleurisy is quite frequent and, in fatal cases, exudations occur in a large proportion shortly before death.

**The Intestines.**—The intestines are only rarely the seat of primary tuberculosis. In children it has been found between 30 and 50 per cent., and in adults Orth and Henke found it in 3 to 5 per cent. of all autopsies. But in phthisis they are secondarily affected to the extent of 90 per cent. of cases, according to some authors. Some of the anatomical changes are merely tuberculous nodules, but in most

there are found round ulcerations of the mucous membrane of the ileus and colon, especially of the ascending colon (Fig. 17). These ulcers heal but rarely, though occasionally there is encountered a case of stricture of the intestine due to a contracted scar resulting from a tuberculous ulcer. On the other hand, these ulcers may perforate into the peritoneal cavity with the usual results of these accidents. Ischio-rectal abscesses are very frequent in phthisical patients.

**Amyloid Degeneration.**—The tuberculous toxemia also causes changes in various other organs which, though not essentially tuberculous, yet are more or less characteristic. Amyloid degeneration occurs mostly in chronic cases of mixed infection. The amyloid material is deposited in the walls of the capillaries outside of the endothelium, and pressing upon the lumen of the vessels, as well as the cells of the organ, prevents the nutrition of the parenchyma. The result is fatty degeneration and atrophy of the organ. We are in the dark as to the origin of this material. The liver, spleen, kidneys and intestines are most frequently affected. Fatty degeneration of the liver is very frequent.

**The Heart.**—In the heart fatty degeneration is usually found in persons who succumb to phthisis. It is usually small, weak and atrophic, as are the rest of the muscles of the body. Hypertrophy of the right heart may be seen in cases of extensive shrinkage of the lung with pleural adhesions. Endocarditis verrucosa is also very frequent, but this is due to streptococci.

**The Muscles.**—The muscles are pale or brown, atrophied and poor in fat. Microscopic examinations show brown atrophy, fatty degeneration and other degenerative changes. It appears that the diminution in the volume of the muscles is due to an atrophy in each individual muscle fiber, and not to diminution in their number.

## CHAPTER VII.

### SYMPTOMATOLOGY OF PHTHISIS—HISTORY OF THE PATIENT.

WE have seen that infection with tubercle bacilli does not invariably result in tuberculous disease. Phthisis implies a preëxisting infection, but the latter may take place without any subsequent clinical manifestation of disease. The diagnosis of tuberculous infection is a simple matter. The application of the cutaneous tuberculin test tells the story promptly, easily, and unequivocally. The chances of error are insignificant and may be disregarded.

But a positive tuberculin reaction, found in over 90 per cent. of humanity, as we have seen above, is by no means proof that the individual suffers from any disease or needs general or special treatment. It only shows that the individual has been infected with tubercle bacilli at some period of his or her life. The infection may not have done any harm. In fact, we have seen that in all probabilities it has immunized him against a new massive infection, which is difficult to avoid and which might have produced acute and progressive disease, had it taken root on virgin soil.

What we aim at in our practice is discovering not only tuberculous infection, but tuberculous disease. At any rate this is what the patient wants to find out: Whether he suffers as a result of the infection with tubercle bacilli and whether any treatment is necessary to save or prolong his life. This information can only be given after a careful and painstaking inquiry into the patient's history, the symptoms he suffers from and the physical signs elicited by an examination of his chest and other parts of his body, and applying some or all the clinical diagnostic methods which have been the achievement of medicine during the past couple of generations.

**Hazards of Hasty Diagnosis.**—Realizing that the patient's chances of recovery are greatest when the disease is recognized and treated at its very incipiency, there has been a strong tendency during recent years to treat every "suspect" as one who is actively tuberculous until time and observation prove the contrary. This advice has been given by many writers on the subject and followed by numerous physicians. As a result many innocent persons have been banished to sanatoriums or to distant climatic resorts; many children have been deprived of an education, many workmen induced to leave their employment, many men of affairs to neglect their business. To be

sure, many of these non-tuberculous individuals—"suspects"—have been fatigued and debilitated and needed a rest, and the error in diagnosis has rather benefited them. But with others things have been different. Many a person known to the writer has been trying to remove the stigma of tuberculosis without avail; and tuberculosis is a stigma at present, despite our teachings that a patient who has common-sense and decency is as good and as harmless as any other person.

We often meet with people who had spent some few months in a sanatorium—from all indications they could have gotten along very well without it—and ever since they live in constant dread lest it will be found out that they had been "consumptives." I have known persons who have lost their jobs because some patient who knew them in an institution "gave them away."

A hasty diagnosis among the poor and moderately well-to-do—from which classes the bulk of phthysical patients are being recruited—works even more havoc at times. The results of the maxim: "Treat everyone for tuberculosis till *he* proves to you that he is not" can be seen in a city like New York where numerous individuals attend tuberculosis clinics for months, even for years, or go from one institution to another for years, though they fail to present any reliable symptoms of active phthisis. I have witnessed the autopsy on the body of a woman who remained twenty-six years continuously in an institution; about one-half the time in a sanatorium, the other half in a hospital for advanced consumptives, where she finally died from pneumonia. Careful examination of the viscera failed to disclose an active tuberculous lesion. I calculated that the community spent, or wasted, over ten thousand dollars on this woman, not including the loss owing to her idleness. We may further mention that during the twenty-six years she kept out of the institution at least forty patients with active disease who might have benefited by the treatment.

Many communities keep on spending considerable sums of money on the maintenance of patients who could be cared for in their homes at a lesser cost, or keep them from work merely because of a suspicion that they are tuberculous. Others break up their homes, commit their children to asylums because of a hasty diagnosis of incipient tuberculosis based on some indefinite symptoms and physical signs. It was found in Germany that some patients, passed for admission to sanatoriums because of incipient tuberculosis, were fit for active military service during the war. Fifty per cent. of patients in one of our largest municipal sanatoriums have negative sputum; that this is an indication that many are non-tuberculous will be agreed to by everyone who has any experience with tuberculosis. With the antiformin method of sputum examination at most 10 per cent. of active cases are found not expectorating bacilli.

There appears to manifest itself a reaction against the eager chase for "incipient" cases which may swell the favorable statistics of



sanatoriums. Authoritative writers now state emphatically that indefinite physical signs should not be relied on, and urge that only constitutional symptoms of toxemia be taken as criteria for active disease. Edward O. Otis<sup>1</sup> questions the wisdom of relying on "the presence of certain physical signs, definite or indefinite, with no symptoms of bacterial toxemia which are interpreted to mean active tuberculosis, and the patient exhibiting such signs is accordingly removed from his family and employment and consigned to a sanatorium, where there is at least some risk that he may receive a new and active infection. Whereas the individual was in no way ill, and probably never would have developed active clinical tuberculosis."

A hasty diagnosis is as dangerous as neglect to recognize active and progressive disease. Delay does not mean sure death of the patient; if he is kept under careful observation we cannot be too late in making a positive diagnosis. The acute and progressive cases will manifest themselves very soon, and delay does not count because treatment in these cases is, as a rule, not very effective. In the slow, sluggish cases the delay of a few weeks hardly ever makes any difference in the ultimate outcome. But pronouncing a patient phthisical when, in fact, he has no symptoms of active disease, is often followed by disastrous results to the patient as well as to those depending on him, and to the community which is charged with caring for its tuberculous dependents. It may be said without fear of meeting contradiction from competent sources that an incipient case in the full sense of the word does not always mean a curable case, or even a favorable case. Many cases justly classed as incipient have a worse prognosis than those considered "far advanced" in the conventional classification of the disease.

**Elementary Principles in the Diagnosis of Active Phthisis.**—Active tuberculosis, or phthisis, manifests itself invariably by symptoms of bacterial intoxication. If there are no symptoms of constitutional toxemia, the patient may have been infected with tubercle bacilli—and who has not been?—but he is not sick with a disease which needs special treatment, costly to the community, and often ruinous to the patient and his family. Nor must the patient be isolated from his family, and hospitalized to prevent the dissemination of a disease which he does not have. This is a point which must always be borne in mind before a patient is told that he suffers from incipient phthisis.

There is hardly a conscientious physician who is not skilled in making a diagnosis of incipient phthisis from the constitutional symptoms, even though he may have to leave the localization of the lesion to some virtuoso in physical diagnosis. There is no active phthisis without fever, cough, tachycardia, languor, nightsweats, hemoptysis, etc. Some or all of these symptoms are found soon after the patient becomes actively phthisical.

<sup>1</sup> New Orleans Med. and Surg. Jour., 1914, lxxvii, 311.

If these elementary points were borne in mind by physicians, the number of mistakes of omission and commission would be reduced to a minimum. In fact, if the propaganda made so assiduously, aggressively and, within certain limits, justly, that to be cured, tuberculosis must be discovered in its incipiency, would have insisted emphatically on the symptomatology of the disease which can be observed and properly interpreted by every practising physician, all cases coming under the observation of physicians would be detected in proper time. It is wrong to blame the general practitioner for the large proportion of cases which are diagnosed rather late, after he has been taught that certain indefinite physical signs may mean phthisis, and just as often may mean nothing. In fact, the general practitioner may retort by saying that the large proportion of non-tuberculous cases admitted and kept in sanatoriums, as well as the large number of patients "cured" within two or three months in the institutions, prove conclusively that the specialists are no less fallible in this regard.

**Natural Method of Arriving at a Diagnosis.**—While in the practice of medicine we must often resort to the deductive method of reasoning when attempting to unravel an obscure case, yet in our attempts at ascertaining the presence or absence of active phthisis, we are on safer ground when applying the inductive method. We must first ascertain the individual symptoms and credit each with its true merit. In other words, all the morbid phenomena must be accurately observed; all the material facts are to be carefully inquired into; and, what is of most importance, the interpretation of the collected facts must be correct and in agreement as regards their relation one to another, and to the probable causes which may underlie the process.

To do this rationally, we must carefully observe the appearance of the patient, go into details about the symptoms which urged him to seek medical advice and also inquire into such subjective symptoms as the average patient is not likely to note unless his attention is drawn to them. When all these data have been carefully gathered and properly evaluated, a physical examination is made to ascertain the objective signs of the disease, as well as an evaluation of the constitutional condition of the patient with a view of ascertaining whether he is endowed with sufficient resistance to counteract the ravages of the disease.

**History of the Patient.**—This is to be minutely inquired into. We find out the condition of health or the cause of death of the patient's parents, and grandparents, if he is in possession of the facts, or capable of giving them to us reliably, which unfortunately is only rarely the case. Of particular importance is whether either of the parents was actively tuberculous when the patient was an infant. In case the parents have become actively tuberculous when the patient had already passed childhood, his chances of becoming phthisical are not greater than of those who do not have such a hereditary taint. In

fact, there appears to be some evidence tending to show that, contrary to the general opinion, tuberculosis, if it occurs at all in such individuals, is apt to run a milder course than in those who have no family history of tuberculosis (see page 112).

We should not be influenced by the age of the patient. No age is immune to the disease, but each age period appears to have its own form of the disease: In infants hematogenic general tuberculosis is the rule; in children tuberculosis of the glands, especially the tracheo-bronchial group, the bones and joints; in adults chronic pulmonary tuberculosis; in persons over forty fibroid phthisis, and in aged individuals a very chronic form with a symptomatology peculiarly its own, etc.

The occupation of the patient has great influences on the chances of developing active phthisis, as was already shown elsewhere, and should be considered when taking the history of the patient. A history of an injury to the chest, especially if followed by hemoptysis, is important.

Preëxisting diseases are to be ascertained in detail. In infants and children active disease is apt to follow in the wake of one of the endemic contagious diseases; in adults, typhoid, pleurisy, pneumonia, diabetes, syphilis, etc., are of etiological moment. A history of scrofula during childhood has very little bearing upon active phthisis in the adult, excepting perhaps that if the disease does occur, it is likely to pursue a mild and exceedingly chronic course. The same is true to a certain extent of previous tuberculous disease of the bones and joints. One has to consider the rarity of old scars on the neck or over joints of phthisical patients; or of phthisis in those who have had Pott's disease during childhood.

In women the menstrual history is to be gone into and special attention paid to amenorrhea. It is also to be borne in mind that active symptoms very often appear immediately after childbirth.

A history of exposure to infection should not be overestimated in adults, as has been advised by many writers. We have seen that those most exposed to infection with tubercle bacilli, as the hospital staffs—doctors, nurses and orderlies—are not more liable to become phthisical than those in other walks of life who do not come into intimate contact with consumptives; nor do the unaffected consorts of tuberculous patients suffer from this disease more than others. It is therefore absurd to expect that working with a tuberculous fellow-workman is more likely to transmit the disease than to a doctor, nurse or unaffected consort. In my own practice I do not at all give exposure to infection any weight in the diagnosis of active phthisis in adults. It is different with children, especially with infants. Infants of tuberculous parentage, or who have otherwise been exposed to infection, are very likely to have contracted the disease in an active form. With children over three we should ascertain whether the parent has become actively tuberculous while the child was less than

one year old, because if the child was older than three years when the parent began to expectorate bacilli, the chances of primary massive infection of the child are remote.

It is a curious fact that, in attempting to trace the source of infection in children, we often find it is one of the grandparents, suffering from senile phthisis, who is responsible, though he or she is ignorant of the true nature of the ailment, having been told that it is bronchitis, emphysema, asthma, etc.

**History of the Present Illness.**—Of immense importance is the history of the mode of onset of the present ailment, as well as certain symptoms from which the patient has suffered during his lifetime. Previous attacks of "grippe," "colds," bronchitis, etc., may mean previous attacks of abortive phthisis and should be carefully considered. The same may be true of typhoid fever, pneumonia, etc., which may have been attacks of acute tuberculosis which have subsided. Having been treated for months for neurasthenia, gastritis, chlorosis, or even malaria is not uncommonly ascertained in the history.

We should inquire into the symptoms which ushered in the present ailment, with special reference to cough, expectoration, lassitude, languor, particularly in the afternoon, loss of weight, hemoptysis, pleuritic pains, or pleurisy with or without effusion, etc. Of most importance in ascertaining the presence or absence of active disease is fever with its concomitant symptoms—chills, backache, anorexia, tachycardia, etc. Night sweats are to be inquired into and it should be ascertained whether they occur immediately upon going to bed, or wake the patient at some time during the night. The appetite of the patient is to be ascertained, and whether any loss in this direction has been concomitant with the appearance of other symptoms. If the patient knows, he should tell the fluctuations in his weight for the past several years. The condition of the bowels, especially the presence of diarrhea is to be ascertained.

Of course, if any sputum is available it should be examined microscopically for tubercle bacilli and chemically for albumin. The urine should be analyzed for the presence or absence of albumin, sugar, and casts.

After all these data have been ascertained we proceed with the physical examination of the patient, and this includes not only a careful examination of the chest by inspection, palpation, percussion and auscultation, but also all other parts of the body from the top of the head to the toes. We may thus find symptoms and signs confirming the diagnosis of phthisis, or proving that the symptoms of which the patient complains are due to some other cause. The stigmata of phthisis are often scattered all over the body, as will be shown later on.

Above all, it must never be lost sight of that while there is no active phthisis without constitutional symptoms, there is no single symptom

or sign pathognomonic of the disease, excepting the expectoration of sputum containing tubercle bacilli, and even this is occasionally apt to mislead. It is only the combination of various symptoms and signs which clinches the diagnosis, especially in obscure cases with negative sputum. This fact by no means interferes with the early recognition of active phthisis, and mistakes are more often due to carelessness in observation than to any other factor.

**Importance of the Symptomatology of Phthisis.**—In the succeeding chapters the physical diagnosis of phthisis in its various forms will be given its proper place, because only with the aid of inspection, percussion, and auscultation can we localize the lesion and gain important hints as to prognosis and the treatment indicated. The symptomatology of the disease, which has been given a subordinate place in some recent treatises on the subject, will be discussed in detail. The reasons are obvious: The general symptomatology of active phthisis can be ascertained by every practising physician and its bearings on the presence or absence of active phthisis, especially in doubtful cases, are of more significance than indefinite physical signs. There may be active phthisis without physical signs revealing themselves even to the best-trained specialist, and many signs of apical involvement are found in healthy persons. But there is no active phthisis without constitutional symptoms. This is an axiom which cannot be repeated too often. The symptomatology of phthisis, when properly studied and interpreted, gives information as to the onset of the disease, its activity, tendency, and ultimate outlook. It can be ascertained by any medical man. Inasmuch as it often precedes the appearance of definite physical signs, or the signs elicited with the aid of skiagraphy, the symptomatology of the disease is to be ascertained first.

We shall therefore begin with a discussion of the most prominent and more or less constant symptoms of active phthisis—cough, expectoration, fever, nightsweats, hemoptysis, anorexia, emaciation, tachycardia, etc. Each of these symptoms will be discussed from the standpoint of diagnosis, differential diagnosis, and prognosis. It is only by a proper appreciation of these symptoms that a diagnosis of active phthisis can be made at any stage of the disease, but especially in the so-called incipient stage; while a prognosis based only on findings during a physical examination and skiagraphy is bound to prove ruinous to any practitioner.

## CHAPTER VIII.

### COUGH AND EXPECTORATION.

#### COUGH.

**Frequency of Cough.**—While cough is the symptom which first attracts the attention of the average patient to his troubles, there has been a question whether there are cases of phthisis without cough. Pidoux stated that cough is the first and last symptom of phthisis; when it is absent, its negative significance is almost absolute. According to many writers, a patient who does not cough is not tuberculous, while there are others who consider it the most constant of symptoms of early phthisis. However, Louis, Wilson Fox, Moeller, and others speak of patients who passed through the disease without ever coughing.

This disagreement is due to various causes. The statement made by many phthysical patients to the effect that they do not cough is to be taken with considerable qualification. Mild cough, clearing the throat in the morning, or hawking, which causes but little annoyance to individuals who are not given to introspection, may be overlooked. Even in the advanced stages, when the patient brings up considerable sputum, there may be no cough—the sputum is carried by the cilia of the bronchial mucous membranes and when it reaches the vocal cords it is easily removed without effort, or swallowed. In the latter case the patient may not even expectorate. I have seen this to be the case with many patients, especially females. For this reason, it is often ascertained by close questioning that there is little, mild cough, “just like everybody else coughs.” I have, however, seen many patients in whom physical exploration of the chest was negative for quite some time, but the continuous cough, productive or unproductive, was the only symptom which urged them to seek a diagnosis, and excited a careful study of the case by the physician.

Another class of patients who do not cough despite active tuberculosis, are aged persons, of whom details will be given later on. The same is true of some cases of phthisis with cavities—mouthfuls of sputum may be brought up without any efforts or cough, as in bronchiectasis.

**Cough in the Early Stage of Phthisis.**—A considerable number of patients give a history of repeated “colds” caught during several preceding winters or autumns; or of attacks of “grippe” which made them cough more or less violently, but they subsided under ordinary treatment. Owing to some neglect, the last attack has been per-

sistent, the cough aggravated and could not be relieved by the remedy which helped them formerly. The cough in these cases is apt to be rather mild, consisting mainly in clearing the throat in the morning and may not at all be productive of sputum; or small lumps of clear, vitreous secretion from the nasopharynx may be brought out. Rarely mucopurulent material is eliminated, but it is usually devoid of tubercle bacilli at this stage.

These repeated attacks of grippe or bronchitis, which subside during the summer to return during the autumn and winter and are easily managed by ordinary sedatives, often give the patient a false sense of security and when told that the cough is of tuberculous origin he is loath to agree to it.

This mild cough is to be differentiated from hysterical cough which is very frequent at present when phthisiophobia is rampant. In fact in many homes with tuberculous patients, most of the healthy members of the family cough, believing they are affected with the disease. Perhaps the best sign is that hysterical cough does not occur at night, when the patient is asleep, or during the day, when he is absorbed in some matter which interests him. I have seen patients who coughed persistently, cease coughing during the time they were engaged in an interesting conversation. The cough in incipient phthisis is annoying at bedtime, disappearing during the first hours of sleep, and reappearing during the early morning hours, often waking the patient, while after rising it may be intense till the chest is cleared. During the day it may be scarce or absent and provoked only by emotional disturbance, undue exertion, chilling the body, a dusty or smoky atmosphere, etc.

**Paroxysmal Cough.**—In many patients at the onset of the disease, or during its later stages, the cough is violent and paroxysmal; occurring in fits. When unproductive it may be difficult to bear because it often increases in intensity during the evening and keeps the patient awake during the night, causing pain in the chest, insomnia, and exhaustion. In others the fits keep up for quite some time till a small piece of viscid mucus is expelled. The first thing these patients ask for is a remedy which will loosen the sputum. During such spells vomiting may occur, or even involuntary evacuation of urine, especially in women with lacerated genitals. The paroxysmal explosions of cough are a frequent cause of hernia in men, especially in those suffering from fibroid phthisis.

Paroxysmal cough in phthisis is said to be due to ulceration of the trachea or its bifurcation. But it is also met with in cases of tracheo-bronchial adenopathy. Its occurrence during periodical evacuation of pulmonary cavities will be discussed later on.

Patients suffering from fibroid phthisis, and those who have tuberculosis evolving in emphysematous lungs suffer at times from severe paroxysms of cough. In these the cyanosis and congestion of the veins of the neck and face are strong features during a paroxysm, and

the suffering may be extreme. The violence of the cough is usually far out of proportion to the amount of sputum brought up. After the expulsion of a small lump of transparent mucus they feel relieved but exhausted, to be annoyed again at longer or shorter intervals. Nocturnal attacks are not uncommon.

I have observed similar paroxysms of violent cough in many cases of galloping consumption in which the lesions were not localized; also in miliary tuberculosis with tubercles widely disseminated all over the lungs and signs of pulmonary emphysema were elicited on physical exploration of the chest. The violence of the cough may be responsible for the extensive dissemination of the tubercles by metastasis. But in many cases under my care the lesion finally localized itself and the disease pursued the usual course of chronic phthisis, the paroxysmal cough disappearing, leaving the common cough encountered in the average case of the disease.

**The Emetic Cough.**—First described by Richard Morton at the end of the seventeenth century, the cough ending in vomiting is quite frequently met with in the early stage of phthisis in various degrees of intensity. Some French authors, notably Paillard,<sup>1</sup> state that the *signe de Morton*, or the *toux émétisante* as they call it, is met with to the extent of 50 to 60 per cent. of all cases of phthisis. This has not been the case with the patients under my care. To be sure, vomiting may be seen in more than one-half the cases of tuberculosis at some period of the course of the disease, but not all vomiting is the result of the true emetic cough, as we shall soon show.

It has been stated that the cough of incipient phthisis often produces no expectoration, but vomiting. There are tuberculous patients who cough as soon as they eat, says Michel Peter,<sup>2</sup> there are others who cough because they eat; there are finally others who, having eaten, cough, vomit and suffer from cardiac palpitation. This cough is so characteristic that when whooping-cough is ruled out, I place great reliance on it in doubtful cases and it has often helped me in making a positive diagnosis sooner than I could have made it without this symptom. But to appreciate its diagnostic significance it must not be confounded with vomiting of other origin which may occur in phthisis. It usually occurs in the following manner:

The patient has had his lunch or dinner with a variable appetite and feels rather satisfied, having no sensation of gastric disturbance, excepting perhaps some feeling of epigastric distention or mild dyspnea. But after the lapse of some time, from five minutes to an hour—an average of about twenty minutes—the patient, either without any warning at all, or feeling some irritation at the back of the throat, is seized with a paroxysm of cough which nearly chokes him; he feels as if he is unable to expel a piece of tenacious mucus which sticks in his throat. Finally he vomits out, in part or completely, the gastric contents which are

<sup>1</sup> La toux émétisante des tuberculeux, Paris, 1911.

<sup>2</sup> Leçons de Clinique médicale, Paris, 1879, ii, 318.



in a variable state of digestion, according to the time they remained in the stomach. There is no sensation of nausea before the paroxysm, but the vomiting comes on suddenly during the coughing spell; a fact which differentiates this form of vomiting from other forms. When occurring for the first time the patient is alarmed, and is inclined to attribute it to some dietetic indiscretion, but if it occurs repeatedly he is compelled to seek another cause. As soon as the vomiting ceases the patient usually feels greatly relieved, the sensation of gastric distention and the dyspnea disappear and at times he may express a desire to eat again. After a time the patient learns prudence from experience—he knows that a heavy meal may bring about a fit of cough followed by vomiting.

During the course of phthisis there occur also other varieties of vomiting which cannot be classified under the heading of the emetic cough. Patients who have been sufferers from chronic gastritis, dilatation of the stomach, and chronic alcoholism often vomit; at times vomiting is provoked by cough. In the advanced stages of the disease vomiting, preceded by cough or not, may occur and in some patients it may be so pronounced as to preclude feeding. But these forms of the vomiting are not the true emetic cough. These patients usually suffer from symptoms of indigestion, furred tongue, foul breath, constipation, diarrhea, headache, etc. Examination usually reveals a dilated stomach, amyloid or fatty degeneration of the liver, symptoms of tuberculous peritonitis, etc. Moreover, while the vomiting may occur after coughing, yet it is not invariably preceded by paroxysmal cough, occurs irregularly, not always after the ingestion of food, and there is no relief immediately after the vomiting. In alcoholics the vomiting is more apt to occur in the morning, and this is also the rule with those in whom the cough is due to chronic pharyngitis. In both these conditions nausea, retching, etc., are common, while in the true emetic cough they are absent. The emetic cough often occurs in the early stages of phthisis, in patients in whom the gastric functions are in good condition, is always preceded by spells of cough, always occurs at a certain time after the ingestion of food, is not preceded nor followed by sensations of nausea, giddiness, faintness, and retching. The reverse, vomiting and then coughing, is never observed.

This form of vomiting, or the emetic cough, is observed in practice in but a few diseases, namely, phthisis, whooping-cough, and in certain forms of pharyngitis. So that when whooping-cough is excluded in a patient with an emetic cough, and the pharynx is found to be in good condition, phthisis is at once to be thought of. If it persists, a diagnosis of tuberculosis may be made even in the absence of definite physical signs of the disease.

Some authors have been inclined to look at the emetic cough as a mechanical accident, comparable with that observed in whooping-cough. But it appears that this does not entirely explain this

phenomenon. If the compression of the abdominal muscles and stomach were the sole cause, we should expect that during violent and prolonged asthmatic paroxysms, vomiting to occur. But I have never seen a patient suffering from asthma vomit after an attack of cough and dyspnea and be relieved immediately after the gastric contents have thus been expelled.

As has been pointed out by Michel Peter, W. Soltau Fenwick,<sup>1</sup> Paillard, and others, the emetic cough appears to be purely a reflex phenomenon, due to irritation by the ingested food of the gastric ends of the vagus and an abnormal excitability of the respiratory centre. Hence, the slightest irritation of the gastric mucous membrane by particles of food is sufficient to produce a violent attack of reflex cough which can bring about vomiting in a mechanical manner.

**Cough during the Advanced Stages of Phthisis.**—With the advance of the disease the cough becomes more and more abundant, more productive, but easier and less exhausting. After the formation of cavities, there is usually observed a diminution in the frequency of the cough, sleep is hardly disturbed during the night when the reflexes are in abeyance and the secretions accumulate in the cavity. But in the morning when compelled to empty the cavities of the secretions there are fits of coughing lasting several minutes, perhaps an hour, and the patient feels relieved.

These patients, like those suffering from bronchiectasis, suffer from cough periodically when the excavations have been filled and need emptying. It may be influenced by posture—as soon as they change their position, the secretions overflow the bronchial tubes and must be brought out by cough which does not cease until all has been discharged. Then there is relief for a variable time until the cavity is again filled. The patients usually learn from experience on which side to sleep if they want to have peace. It is not always on the healthy side on which they can lie with more or less comfort, because, like in bronchiectasis, it depends on the direction of the bronchus or sinus which empties the cavity. Patients with pleural effusions also cough when changing their positions, but in their case the cough is usually dry and is not instrumental in bringing up abundant sputum. For obvious reasons, patients cough more when lying down than when in the upright position.

In some cases the cough at this stage is very severe and almost incessant, painful, and preventing rest day and night; actually exhausting. It is noteworthy that the severity of the cough does not altogether depend on the extent of the lesion in the lung, nor on the size and number of the cavities. Some will cough very little, although the lungs are extensively involved, while others, with limited infiltrations or excavations, cough severely.

The cough of tuberculous patients is often greatly influenced by

<sup>1</sup> *The Dyspepsia of Phthisis*, London, 1894, p. 118.

various factors, of which the age and the emotional state are most important. Young adults cough, as a rule, more than old consumptives. In fact, a large proportion of old people suffering from phthisis hardly cough; they bring up large quantities of sputum without any effort. They are the patients who supply the material for those who describe cases which have been sick with the disease for many years and never coughed. The psychic state of the patient also has a great influence. The nervous, irritable, and hysterical cough more than the indolent and phlegmatic. The former class is also more apt to suffer from the emetic form of cough.

**Diagnostic and Prognostic Significance.**—On the whole, cough serves a very good purpose by drawing the attention of many patients to the condition of their lungs. A person who never coughed, but “caught cold” for the first time after his twentieth year, and as a result keeps on coughing for more than a month, is to be strongly suspected of being tuberculous, even if there are no definite physical signs of a pulmonary lesion. The suspicion is fortified by a history of the absence of acute coryza during the first few days of illness because simple bronchitis and “grippe” are almost always preceded or accompanied by nasopharyngeal catarrh.

From the prognostic viewpoint cough is important because we meet cases with small pulmonary foci without much fever, anorexia, emaciation, etc., who would undoubtedly do well but for a cough which is difficult to control. If violent, paroxysmal, and continuing for some time, the cough may be instrumental in extending the lesion, exhausting the patient and thus aggravating the outlook. It also irritates the larynx, trachea, bronchi, and pulmonary parenchyma, and predisposes these organs to infection by metastasis of the bacilli. Violent fits of cough may also be responsible for spontaneous pneumothorax in cases in which the lesion is located superficially or subpleurally. Kuthy and Wolff-Eisner<sup>1</sup> say that the most unfavorable prognosis is to be given in cases in which the patient coughs during both day and night; relatively more favorable is the outlook when he coughs during the day exclusively; more favorable when he coughs only mornings and evenings; and most favorable when he coughs exclusively in the morning.

Within certain limits cough also gives other prognostic hints. With each improvement in the local or general condition the cough also improves or disappears, and with every recrudescence of cough we may find an extension of the process in the lungs, or some complication in the bronchi or nasopharynx. Occasionally we may note that the sudden disappearance of cough is a signal of some grave complication of phthisis, especially meningitis or peritonitis. The same is at times seen in cases of severe ulcerations of the larynx, causing dysphagia, etc. The cough may be ameliorated, but the lesion in the lungs

<sup>1</sup> Die Prognosenstellung bei der Lungentuberkulose, Berlin, 1914, p. 219.

continues or extends and, combined with the exhaustion due to lack of nourishment, the end is not very far.

**Hoarseness.**—Changes in the timbre of the voice may appear quite early in the disease without any tuberculous involvement of the larynx. The least provocation, such as changes in the weather, or prolonged speaking, may produce dysphonia, or a muffled voice without any pain which, with the dyspnea preventing speaking continuously long sentences, may be quite troublesome.

In many cases the hoarseness is due to simple catarrh caused by chemical irritation of the larynx by the secretions while they are being eliminated from the lungs. In others, pressure of a tuberculous gland, lying between the trachea and the esophagus, on the recurrent laryngeal nerve causing adductor paralysis, is the cause. Often the hoarseness is due to tenacious secretions sticking to the vocal cords, and after coughing strongly they are dislodged and the voice is again normal. Congestion of the larynx caused by violent fits of coughing may be the reason for hoarseness.

It is thus evident that not all cases of hoarseness, or even dysphagia, are due to tuberculous ulcerations of the larynx. In fact, no diagnosis of the latter condition should be made without a careful and painstaking inspection of the larynx with a mirror.

### EXPECTORATION.

Careful inquiry reveals in most cases that the cough preceded expectoration by several weeks or even months, and we must not unequivocally conclude that because the cough is unproductive we are not dealing with phthisis. Children before the sixth year never bring up any sputum at all, because they unconsciously swallow it, and most women do the same. I have met with cases in which urging women to expectorate was of no avail. Many men are not much better in this regard and for reasons of false delicacy they swallow the sputum, especially during the early stages of the disease. In the advanced stage we may meet with the same condition when the patient is exhausted and hardly has any strength to rise or turn around in bed and expectorate into the sputum cup.

With the advance of the disease the quantity of sputum eliminated increases, but I have met with cases showing extensive infiltrations of more than one lobe, without any substantial expectoration, and in some of these I have been convinced that they had not swallowed the sputum. It was merely an indication that the tubercles had not broken through a bronchus.

**Macroscopic Appearance of the Sputum.**—There is nothing typical about the naked-eye appearance of the sputum in early phthisis, although ancient clinicians gave detailed descriptions of typical tuberculous sputum. Perhaps the fact that they knew very little

about early phthisis will account for their confidence in the gross appearance of the sputum in this disease.

In the early stages we find that the sputum is scanty; at times it is altogether absent. Kuthy found that in 49 per cent. of cases in the first stage, 15.4 per cent. of the second stage, and 12 per cent. of the third stage, sputum was altogether absent. What is usually brought up in the early stages is viscid mucus, occasionally with some dark points; it is often frothy and floats on water, hardly differing from the expectoration in bronchitis.

With the advance of the disease the sputum becomes thicker, although it remains glassy or transparent for some time, but yellow streaks are to be seen indicating that it is assuming a purulent character. Later its appearance and consistency change: It becomes mucopurulent and finally purulent, indicating that softening of lung tissue has taken place and the necrotic parts are being eliminated. The purulent character of the expectoration is judged by the yellow, yellowish-green, or green color it assumes. Pure purulent sputum without froth is mostly seen in cases in which an abscess or pyopneumothorax has broken through a bronchus.

In the far-advanced stage of the disease the sputum is usually dark gray or greenish in color, made up of roundish balls which float around like islands in the fluid mucus or saliva or, when thicker in consistency, sink down to the bottom of the receptacle where it settles in disk or coin-shaped masses which keep apart and do not coalesce. This is the nummular sputum of old physicians which had erroneously been considered pathognomonic of phthisical excavations. At times whitish, cheesy masses, derived from broken-down tubercles, are seen scattered within this sputum.

This sputum is usually odorless, but at times it acquires a very disagreeable, sweetish, but nauseating odor, especially when retained within the chest by narcotic drugs, or weakness of the patient. Fetid sputum of this character is exceedingly rare in phthisis. Whenever it is met with we should look for complicating pulmonary gangrene, which occurs at times. Very rarely it is due to fetid bronchitis. It is usually salty in the early stages but later it often acquires a sweetish, sickening taste.

Very often this sputum, derived from tuberculous cavities, when allowed to stand in a vessel for some hours separates into three layers—an upper frothy layer; a middle thin serous layer; and a lower layer consisting of thick plugs of pus. This is characteristic of excavation but not of necessarily tuberculous origin. Bronchiectasis, and also chronic bronchitis with copious expectoration may also be productive of sputum which separates on standing. However, in the former the lines of demarcation between the layers are not as distinct, but one passes into the other by slow gradations.

There are cases of advanced chronic phthisis with scanty, or even without any expectoration, especially those of the types of fibroid

phthisis or with emphysema, although they have periods in which the expectoration is quite profuse. The expectoration decreases in quantity when the cavities "dry up" during the process of healing, and in other cases when the concomitant bronchitis disappears. With but few exceptions, scanty expectoration speaks for a favorable outlook, provided the cough is also absent or mild. On the other hand, copious expectoration *per se* is not always an unfavorable sign. It is an indication of excavation, bronchitis or bronchiectasis which are not infrequent in phthisis. In the latter cases the sputum may show a tendency to collect and be expelled at intervals in very large quantities—mouthfuls—without any effort, and may also be influenced by posture. Of course, in pyopneumothorax breaking through the lung, profuse expectoration of purulent material is seen.

During hemoptysis the material expectorated is sanguinous in various degrees, corresponding to the severity of the bleeding, and for a few days after the cessation of the active hemorrhage the sputum contains dark clots derived from the blood that has coagulated in the bronchi and is being slowly eliminated. The sputum may have a reddish or chocolate tinge without distinct hemorrhage, and even rusty sputum characteristic of pneumonia is at times encountered in phthisis. Inasmuch as this is, as a rule, seen during an acute exacerbation of fever, etc., I am at times inclined to account for it by intercurrent pneumonia. In some advanced cases I have seen at the terminal stage thin, watery sputum, dark brown in color, with numerous air bubbles—prune-juice sputum—which is an indication of pulmonary edema. Green sputum is at times met with, and is usually ascribed to, the implantation of the *Bacillus pyocyaneus*. In cases in which a pyopneumothorax communicates with a bronchus, as well as when an empyema breaks through a bronchus, the sputum may be distinctly purulent, and I have seen cases in which the empyema was thus cured, though the tuberculous process went on its course.

### EXAMINATION OF THE SPUTUM.

**Collection of Specimen.**—In cases of suspected phthisis the sputum gives important information which is often of more value than all other diagnostic methods for this disease. This is especially true of the microscopic examination, and to a certain extent of the chemical examination.

It is important, especially in cases with scanty expectoration, that the specimen of sputum for examination should be properly collected. The patient must be warned that what we want is material that has been coughed up from beneath the glottis, and not what has been hawked out from the nasopharynx, or saliva. A clean, wide-mouthed bottle is the best receptacle, and it should be tightly corked. The one used by the Health Department in New York City is excellent. In cases with scanty expectoration, a twenty-four-hour specimen is

desirable, but with others the quantity coughed up during the morning on rising is sufficient. Fresh sputum is best, but putrefaction does not interfere with the appearance of the bacilli under the microscope.

**Microscopic Examination.**—In incipient cases tubercle bacilli are more often absent than present in the sputum, and it is only when softening of tubercles has taken place and the diseased focus opens into a bronchiole that they can be found. In general, it may be stated that severe cases show large numbers of bacilli, but there are many exceptions. In fact, in acute pneumonic phthisis bacilli are often lacking. The absence of bacilli is therefore not conclusive proof of the non-tuberculous character of a case, because we meet with undoubted cases of tuberculosis, proved by subsequent autopsy findings, in which no bacilli were discovered throughout the course of the disease. In general, it may, however, be stated that these "closed" cases of tuberculosis run a more favorable course. On the other hand, in acute miliary tuberculosis, tubercle bacilli are exceedingly rare.

In early phthisis in which it is difficult to obtain sufficient sputum for examination, the administration of iodides, 5 grains three times a day for a couple of days, may increase the amount of expectoration. We may in some cases also administer an opiate in the evening with a view of retaining the sputum during the night, so that it may be brought up in the morning on rising. In children, swabbing the throat with some gauze, as suggested by Holt, may yield a specimen for examination.

**Technic.**—The examination is best and most rapidly accomplished by the Ziehl-Neelsen, the Gabbet, or the Hermann methods, which have survived numerous modifications introduced during recent years.

With a platinum-wire loop a cheesy or mucopurulent particle is picked out and spread over a perfectly clean cover-glass in a thin, uniform layer. It is even better that a small amount of sputum should be spread between two cover-glasses which are drawn apart. The cover-glass is dried in the air or over a Bunsen burner at some distance from the flame. When dry it is "fixed" by passing it three or four times through the flame. Some of the solution (carbol-fuchsin, 1; absolute alcohol, 10; carbolic acid, 5; and distilled water ad. 100) is put on the specimen which is picked up with a Cornet forceps and held over the flame for about three minutes or more till it steams, or bubbles appear over it. It is then decolorized in a 10 per cent. solution of nitric acid, or a 30 per cent. solution of sulphuric acid and washed in 60 per cent. alcohol till it is completely colorless, when it is counterstained with an alcoholic solution of methylene blue, washed in water and dried between filter paper.

With Gabbet's method the staining with carbol-fuchsin is the same as above, but the decolorization and counterstaining are done together by placing the specimen in Gabbet's solution (methylene blue, 2; sulphuric acid, 25; distilled water, 75).

The Hermann stain is also easy; it consists in: (a) Crystal violet, 3 per cent. in alcohol; (b) ammonium carbonate, 1 per cent. solution in water. Mix one part of solution *a* with three parts of solution *b* just before using. Steam as above, decolorize with 10 per cent. nitric acid, wash in alcohol, and counterstain with Bismarck brown. At times this method will reveal bacilli when the above have failed.

These methods will disclose the bacilli in the vast majority of cases, but they fail at times because of the small amount of sputum available, or the small number of bacilli present in the specimen, or the selection of a particle of sputum with the platinum loop which does not contain any bacilli. To obviate these sources of error there have been devised new methods which liquefy the sputum, digest all the cells and bacteria which may be present, excepting the tubercle bacilli, which can be centrifuged and be examined microscopically, and may even be used for cultural purposes or for injections into animals. The antiformin method is at present the best and simplest available for the purpose.

**The Antiformin Method.**—Devised by Uhlenhuth and Xylander, and modified by others, this method consists in mixing the sputum with antiformin—a strongly alkaline mixture of sodium hypochlorite, equivalent to 5.68 gms. available chlorine; sodium hydroxide, 7.8 gms., and sodium carbonate, 0.32 gm.—used by brewers in the disinfection of their fermentation vats and tubes. When properly diluted and mixed with sputum, there is a strong liberation of gas, the insoluble organic matters, as well as bacteria, are destroyed, excepting hair, fat, wax and cellulose, and acid-fast bacilli, the vitality and staining reactions of which remain unchanged. The resulting yellowish solution is a homogeneous mixture with a flocculent sediment. Because it has a fatty capsule the tubercle bacillus remains intact while all other microorganisms are rapidly destroyed.

Of the various modifications of Uhlenhuth's original method, the one devised by Boardman<sup>1</sup> is the most serviceable. It consists in:

1. Place the entire twenty-four-hours' sputum in a conical settling glass; if the amount is excessive it is perhaps better to use only 15 to 20 c.c.

2. If the specimen is thick add an equal volume of distilled water. Less tenacious specimens do not require so much dilution.

3. Add an amount of antiformin equal to one-fourth the volume of the diluted sputum; in other words, sufficient to make a 20 per cent. solution.

4. Stir thoroughly, thereby breaking up the masses of mucus and greatly hastening complete solution.

5. Allow to stand till solution appears homogeneous. It should now be watery in consistency and pale yellow in color; if necessary, more water or more antiformin should be added and digestion allowed

<sup>1</sup> Johns Hopkins Hosp. Bull., 1911, xxii, 269.



to continue. This will usually require from a few minutes to an hour but may be allowed to continue for days with no resulting harm to the tubercle bacilli.

6. Add an equal volume of 95 per cent. alcohol. By this procedure the specific gravity is reduced from about 1.030 to below 1; thereby not only hastening sedimentation, but making it more complete.

7. After stirring, allow to stand till sedimentation is complete. This will occur in from two to four hours, but a period of twelve to twenty-four hours is recommended. During this sedimentation it may be necessary to gently turn the vessel to dislodge little particles of sediment which may be adhering to the sides of the vessel.

8. Pour off the clear supernatant fluid.

9. Make smear from sediment on a glass slide, using some of the original sputum to aid in fixing the smear. This is best done by making a smear from the sputum before antiformin is added and afterward spreading the sediment from the sputum-antiformin mixture on the same slide. Stain in the usual way.

There are many modifications of this method which do not require twenty-four hours for execution. Loeffler's modification, which takes but ten minutes is the best:

A certain quantity of sputum (10 to 20 c.c.) is mixed with an equal quantity of 50 per cent. aqueous solution of antiformin and boiled over the flame. Rapid liquefaction is observed. To each 10 c.c. of the mixture, 1.5 c.c. of a 10 per cent. alcoholic solution of chloroform is added. After stirring for some time the solution is centrifuged for about fifteen minutes. The disk which forms on the surface of the chloroform contains the tubercle bacilli, and is to be pipetted, fixed with egg albumen and stained in the usual way.

The great importance of the antiformin method lies in the fact that it exerts a destructive action on all cells and microorganisms excepting the acid-fast rods which may then be found microscopically. But soon after its introduction it was found that the acid-fast rods which are not pathogenic, and which are often found while looking for tubercle bacilli, may escape destruction by the antiformin thus causing mistakes. Especially was the question whether the smegma bacillus is dissolved by this agent important. In a recent investigation of this problem by von Spindler-Engelsen,<sup>1</sup> she found that the smegma, the timothy-hay bacillus, the butter bacillus, etc., are dissolved by 15 per cent. of antiformin in thirty minutes. The human and the bovine types of tubercle bacilli were not affected with a 50 per cent. antiformin solution for four days. Under the circumstances it appears that the pathogenic bacteria may be discovered with the aid of this method. It is, however, important that a fresh solution of antiformin should always be used, because a weak and old solution may leave the non-pathogenic bacteria and thus lead to error.

<sup>1</sup> Centralblatt f. Bakteriologie, 1915, lxxvi, 356.

**Much's Granules.**—As has already been stated there are cases of pulmonary tuberculosis in which no acid-fast bacilli can be discovered in the sputum by any method, and Much has shown that they are due to a certain kind of bacilli which have lost their acid-fast property, but are Gram-positive and they retain their virulence. According to some authors these Much granules are almost always found in cases of fibroid phthisis, chronic bronchitis, emphysema, bronchiectasis, etc., in which acid-fast bacilli are very rarely discovered (see p. 18). Much found them in cases of cold abscess.

As to the causes why the bacilli lose their acid-fast properties, there is no agreement. It also appears that the proportion of cases in which they are found varies with different observers, some having detected them in as many as one out of eight sputa, while others in less than 2 per cent. Much gives several methods for staining these granules. The following is the most suitable:

A very thin smear is made of the sputum and allowed to remain for twenty-four to forty-eight hours in a methyl-violet solution (methyl-violet, 10 c.c. of a saturated solution, in 100 c.c. of a 2 per cent. watery solution of carbolic acid) at 37° C. temperature; or it may be stained by boiling for a few minutes over the flame. Wash and stain for one to five minutes with Gram's iodine and put for one minute in a 5 per cent. nitric acid solution, then in a 3 per cent. hydrochloric acid solution for ten seconds, and finally complete the decolorization by placing it for a few seconds in acetone-alcohol (equal parts of acetone and alcohol). Wash and dry.

**Prognostic Value of Microscopic Findings.**—The interest displayed by many patients, as well as by physicians, in the number of bacilli found in a specimen of sputum examined with a view of drawing prognostic conclusions is unjustified. There are cases which show but few bacilli in each specimen, yet they run a very acute and progressive course, while others with numerous bacilli pursue a slow, chronic course, terminating in recovery. Especially is this seen in senile phthisis, in which the number of bacilli expectorated is enormous and we may, in fact, speak of pure cultures; yet these "bacilli carriers" live on for years with comparative comfort. Of course, in such cases we may deal with a small ulcerating cavity in the lung which offers good opportunities for the growth of bacilli, but the fibrous capsule prevents the extension of the lesion.

The number of bacilli in the sputum fluctuates from day to day, evidently depending to some extent on the bit of sputum we happen to pick up with the loop. On the other hand, the complete absence of bacilli from the sputum for several weeks, coupled with improvement in the general condition of the patient, is undoubtedly a favorable sign. But many chronic cases, especially fibroid phthisis, are always "closed"—bacilli are scanty or absent. With modern methods of antiformin examination of sputum the number of "closed" cases have been reduced very much.

**Inoculation.**—In very suspicious cases in which a diagnosis is imperative, but the microscopic findings are negative, inoculation of the sputum into guinea-pigs may clear up the case. The simplest way is to inject it subcutaneously by means of a hypodermic syringe; or a pocket is made by a small incision and the sputum introduced with a platinum loop. The best place is the abdomen. After three weeks the animal is examined for enlargement of the regional lymphatic glands. If these are not found enlarged, the guinea-pig is killed after waiting two months, and if suspicious areas are found at autopsy they are examined carefully. In most cases the regional lymph glands are enlarged in four or five weeks to the size of a pea and palpable. The animals may then be killed with chloroform with a view of more careful examination at the autopsy.

There are, however, on rare occasions cases in which it is of great importance to ascertain the presence or absence of tubercle bacilli in the sputum sooner than in six or eight weeks. Some have suggested that after the suspected material has been injected into the abdominal wall or the peritoneum, the animal should be tested at frequent intervals with tuberculin. A positive reaction clears up the case (Römer and Joseph).<sup>1</sup> Martin Jacoby and N. Meyer<sup>2</sup> suggest that the sputum be injected into a guinea-pig and about fourteen days later 0.5 c.c. of tuberculin should be injected subcutaneously. If the sputum contains tubercle bacilli and infects the animal, it will die from anaphylactic shock within a few hours.

**Elastic Fibers.**—Before the discovery of the tubercle bacillus great stress was laid on the presence or absence of elastic tissue in the sputum in the diagnosis of tuberculosis, but of late this is only rarely looked for. It is, however, a simple thing to find elastic tissue when present in the expectoration, and it is of immense diagnostic significance because it can be found in over 90 per cent. of tuberculous sputa.

The presence of elastic fibers in the sputum is an indication of destruction of lung tissue and it may be found in the very early stages of the disease, because chronic tuberculosis is a destructive process and small excavations may be found quite early and the elastic fibers are not destroyed during the caseous degeneration which liquefies the pulmonary tissue. It is also found in gangrene, abscess, syphilis and infarction of the lung, so that when the latter can be excluded, it may greatly assist in the diagnosis of doubtful cases of tuberculosis.

**Technic.**—A small amount of the thick purulent portion of the sputum is pressed into a thin layer between two pieces of plain window-glass, 15 x 15 cm. and 10 x 10 cm. The particles of elastic tissue appear on a black background as grayish-yellow spots, and can be examined *in situ* under a low power. Or, the upper piece of glass is slid off till the piece of tissue is uncovered, when it is picked out and

<sup>1</sup> Beitr. z. Klinik d. Tuberkulose, 1909, xiv, 1.

<sup>2</sup> Ibid., 1911, xx, 263.

examined on a slide, first with a low and then with a high power (Simon).

A simpler method is the following: A bit of purulent sputum and a drop of 10 per cent. solution of sodium or potassium hydrate are placed between a cover-glass and a slide and examined with a moderate power under the microscope. The elastic tissue is to be looked for especially at the border of the preparation.

If the fibers are scanty they may not be found in this way, and the following method may reveal them: The sputum is boiled with a 10 per cent. solution of KHO and well stirred during the boiling. When a homogeneous mixture is obtained it is diluted with four times as much water, well shaken, and allowed to stand in a conical glass, or centrifuged. The sediment contains all the elastic tissue which may be found under the microscope.

The different methods of staining elastic tissue are not necessary because either of the above methods is sufficient for diagnostic purposes.

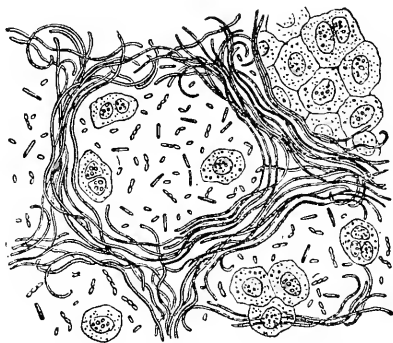


FIG. 20.—Elastic fibers in the sputum. (v. Jaksch.)

**Cytology of Sputum.**—Various attempts have been made to assign diagnostic and prognostic significance to the cytology of tuberculous sputum, especially to the leukocytes and lymphocytes, but without avail. Nothing diagnostically important can be learned from a study of the white-blood cells in the sputum, as far as we know at present.

**Chemical Examination.**—The chemistry of the sputum in pulmonary tuberculosis has not yielded any important diagnostic or prognostic data, excepting the albumin reaction which is of immense value in doubtful cases and is often of assistance when the microscope fails to reveal tubercle bacilli. Sputum with a positive albumin reaction can be found in tuberculosis and also in cases of pulmonary emphysema with cardiac dilatation, pneumonia, pleurisy with effusion, etc., but never in uncomplicated bronchitis.

A positive albumin reaction is not always decisive of tuberculosis, but the negative outcome, when persistent during several examina-

tions, undoubtedly excludes phthisis.<sup>1</sup> In some cases of advanced tuberculosis, especially fibroid phthisis, the albumin reaction is negative, but in such cases the diagnosis is only rarely a problem. It also appears that with the improvement in the condition of the average patient, the amount of albumin in the sputum decreases and finally it disappears. It is thus of prognostic value.

**Technic.**—The albumin test is made as follows: A 3 per cent. solution of acetic acid is added to the sputum, which is then thoroughly shaken. During ten or fifteen minutes the bottle is allowed to stand, and repeatedly shaken during this time. It will be observed that the mucus is coagulated by the acetic acid, and when it is then filtered through paper into a test tube, the filtrate appears as a clear fluid. Occasionally all the mucus is not coagulated with the first attempt and this is easily ascertained by adding a drop of acetic acid to the filtrate, which in such cases again shows flocculi collecting as a precipitate. The process is then repeated until a clear filtrate is obtained. The clear fluid is next boiled over a Bunsen burner or an alcohol lamp and while boiling some crystals of common salt, or a concentrated solution of sodium chloride, are added.

If albumin is present, there results a cloudiness, or a curdy precipitate which, on standing, settles to the bottom of the tube. Roughly speaking, the amount of the precipitate gives an idea of the amount of albumin present. The most important precaution to be observed is that nothing but a curdy precipitate should be considered as positive, because the presence of mucus, which the acetic acid does not always completely dissolve, may also give a cloudy precipitate on boiling. But this reaction is not curdy, nor does it settle on standing. Of course, any other test for albumin may be used on the filtrate, but the above gives satisfactory results.

<sup>1</sup> Fishberg, *Med. Press and Circular*, 1912, xciv, 352; *Arch. of Diag.*, 1912, v, 220.

## CHAPTER IX.

### FEVER AND NIGHTSWEATS.

#### FEVER.

FEVER is one of the first symptoms of active phthisis—perhaps the first. It does not run a characteristic course in every case like that in malaria, pneumonia or typhoid fever; in fact, its polymorphism is noteworthy. Yet it is of immense diagnostic and prognostic value. Some authors state that the fever in incipient tuberculosis is invariably due to some complication. But the tuberculin reaction, as well as acute miliary tuberculosis show clearly that this view is incorrect. All the available evidence combines to prove that it is due to absorption of the poisons produced by the tubercle bacilli, though it may be modified by mixed infections. The fever is engendered mainly by the increased production of heat—the result of complex chemical processes having their origin in the struggle of the organism with the bacilli; the body summoning its defensive forces against the toxins produced by the decaying tissues. These latter stimulate the heat regulating centre. This is confirmed by the fact that nervous individuals are more apt to have fever than others, and after mental excitement the fever often rises in the tuberculous patient. In evaluating the significance of fever in tuberculosis it must be borne in mind that it is not the cause of the disease, but a result of its activity.

Fever is present in nearly all cases of active disease. In the later stages, especially in fibroid phthisis, we often meet with afebrile periods of shorter or longer duration, but with each exacerbation of the disease, with each extension of the process in the lungs there is always a pronounced rise in the temperature which should be studied if the evolution of the case is to be followed.

**Thermometers.**—The reason why there are found so many apyretic cases of phthisis is mainly faulty technic in taking the temperature, especially defective thermometers.

The clinical thermometer is an instrument of precision and when used for the purpose of ascertaining the temperature in incipient phthisis, in which 1° is occasionally of immense importance in diagnosis and prognosis, it must be accurate. It is, however, a well-known fact that, despite the certified accuracy of each instrument, simultaneous observations made on a single patient with two instruments often disclose a difference in readings of 0.75° to 2°. The simultaneous immersion of two dozen thermometers in a bath of warm water disclosed that the readings varied from 98.2° to 101.6° F.; another

similar batch of higher-priced thermometers in another bath showed variations of temperature between 98° and 105.4° F.<sup>1</sup> "Certified" thermometers in this country are not much better. Bray<sup>2</sup> reports that out of a series of 83 certified thermometers tested in a water-bath, 17 showed a variation of 0.3° to 0.6° F. Comparative rectal readings approximated closely the discrepancies shown in the water-bath. The presence or absence of fever, when such thermometers are used to ascertain it, depends on the instrument which the physician happens to possess and not at all on the condition of the patient. Under the circumstances, it is clear that when searching for fever in tuberculous patients or suspects, the instruments must be reliable and of tested accuracy, otherwise grave diagnostic mistakes of omission or commission are likely to occur.

**Technic of Taking the Temperature.**—After having a good thermometer, we must exercise great care in the method of taking the temperature. I have been so often misled by readings taken in the axilla, sometimes finding it as much as 3° below that recorded in the rectum, that I now completely discard it. And, strange to say, I meet with no patients who refuse to take their temperature per rectum. It has been found that in some cases the temperature in the axilla is higher on the affected side and urged as a good sign of phthisis, but it is so rare that it may be disregarded.

The mouth temperature is also unreliable to a certain extent. Here it is influenced by the temperature of the external air which must be inhaled now and then, especially by patients suffering from nasal obstruction. The part of the instrument outside the lips, and at times also the part within the mouth, are chilled by the external air, more often in dyspneic patients. The instrument must be left in the mouth at least seven minutes, and it often takes at least ten minutes before the mercury rises to the highest point, even with the so-called "minute thermometers." On the other hand, in patients suffering from stomatitis, the local temperature may be much higher than that of the blood. The temperature in the mouth should also not be taken immediately after meals, after taking hot or cold drinks, after washing the mouth or brushing the teeth, etc. Many patients also are unable to keep the thermometer properly beneath the tongue, all surrounded by buccal mucous membrane, and avoid breathing through the mouth or talking for five to ten minutes.

It appears that the majority of physicians in sanatoriums are in favor of oral readings because they are dealing with patients who practically always associate in groups and cannot use the rectal method unless they retire to their rooms for the purpose several times a day. This drawback does not hold with bed-ridden patients, and also with the average clientele in the city. In fact, I found that suspects, who keep at their work while under medical observation,

<sup>1</sup> *Lancet*, October 4, 1913; November 8, 1913, p. 1342.

<sup>2</sup> *Amer. Jour. Med. Sci.*, 1915, cxliv, 838.

prefer the rectal method which they take in the lavatory and thus obviate observation by others. In my hospital work also, there is no trouble in taking rectal temperature in walking patients.

That the rectal method is superior and less likely to mislead is now acknowledged by all who have given both methods a trial. In the rectum or vagina the instrument is on all sides surrounded by mucous membrane, holding it in place as long as necessary and giving reliable readings. It has been found that the rectal is almost invariably 0.5° to 1° F. higher than the mouth temperature (Fig. 21). It is needless to add that the instrument is to be left in the rectum sufficiently long to obtain the maximum reading. In my instructions to patients and nurses, I tell them that I do not know of any one-minute thermometer, and all are to be left *in situ* at least five minutes.

**Frequency of Taking the Temperature.**—The habit of many physicians of taking the temperature when the patient visits them and

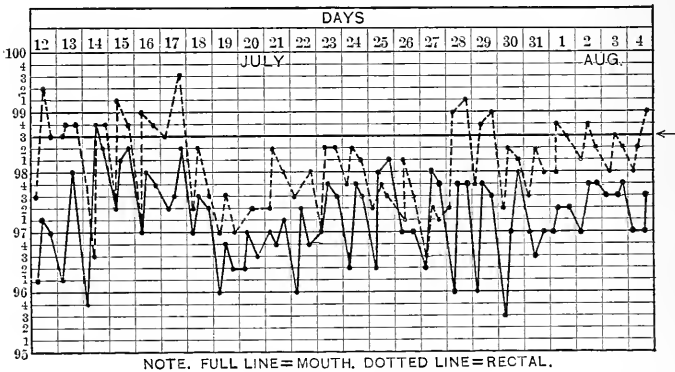


FIG. 21.—Comparative oral and rectal readings of temperature. (Bray.)

recording it as normal or elevated to a certain degree is altogether wrong. In incipient or doubtful cases taking three readings a day may be misleading at times because rises in temperature which occur late at night or early in the afternoon, and are short-lived, may thus be overlooked and the patient pronounced free from fever. For reasons which will soon be evident, we must, in incipient cases, have a record of the temperature taken every two hours, and this is best recorded by plotting a curve on a chart which shows graphically any hypothermia or hyperthermia.

Intelligent patients may be entrusted with a thermometer, provided they are trained in reading it correctly, which can be done in a few minutes. I have had patients who kept records of their two-hourly temperature for weeks and, for obvious reasons, more conscientiously than the average nurse. Many have done it without leaving their occupations by simply going to the lavatory every two hours for five minutes.



**The Normal Temperature.**—It may be stated that the normal temperature in children is not a constant value. It is subject to such oscillations during perfect health, that any average which has been fixed by various authors is only arbitrary. The slightest disturbance in health is likely to increase the temperature in the child to a greater degree than in the adult. Many clinicians consider a temperature of 100° to 101° F. normal in a child, unless there are symptoms of disease. But with advancing age the temperature becomes more and more settled, so that in adults it is subject to lesser oscillations, unless raised or depressed by disease.

As an arbitrary guide for the clinician it may be taken that a temperature of 98.6° F. when taken by mouth, and 0.5° higher when taken by the rectum, is normal. But even this shows striking diurnal variations in normal individuals. During the early morning hours, before the individual leaves his bed, it is slightly subnormal from 0.5° to 1°; but it rises to normal soon after rising, and keeps quite steady

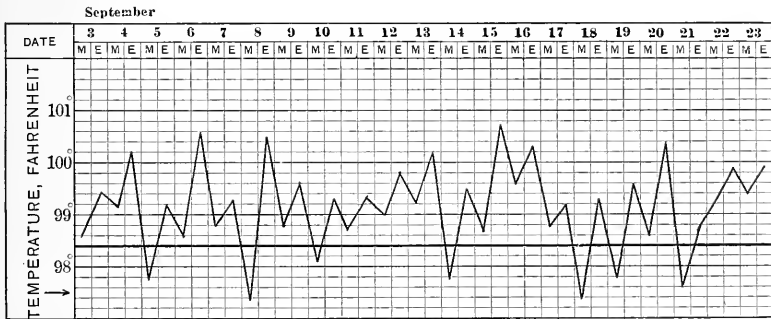


FIG. 22.—Fever in incipient tuberculosis showing marked subnormal temperature in the early morning hours. Temperature taken twice daily.

during the day. Bardswell and Chapman<sup>1</sup> found an average for waking hours 98.5° F., and for sleeping hours 97.2° F., taken by mouth, which is in agreement with the observations of most physicians.

There are, however, individuals in whom the temperature is lower than the above average and in whom a physiological normal temperature should be considered febrile. This is occasionally seen in tuberculous patients with subnormal temperature; when the thermometer registers 99° F. they present symptoms of fever, such as flushing, hot skin, headache, etc.

Normally the temperature is elevated in persons after exercise, and in some even after a hearty meal. In women it may be higher by 1° or 2° before or during menstruation. But the elevation after exercise is, in the healthy individual, evanescent; within one-half to one hour it sinks again to normal.

<sup>1</sup> Brit. Med. Jour., 1911, i, 1106.

Other influences which should be mentioned are the emotional state of the individual. Particularly in women, excitement may raise the temperature  $1^{\circ}$  to  $2^{\circ}$ . Where there is a question of tuberculosis, the excitement attending the taking the temperature may be effective in raising it, as I have seen in several cases, and we must be very careful in making a diagnosis of incipient phthisis on the thermometrical readings alone in emotional women.

In some people who work during the night and sleep during the day, the variations in temperature mentioned above are said to be reversed.

In evaluating thermometrical findings in suspected incipient phthisis we are on safe ground when we consider the normal temperature during the day in a person who works or walks around as  $99^{\circ}$  F. when taken per rectum, and  $0.5^{\circ}$  to  $0.75^{\circ}$  lower when taken by mouth. It may be  $0.5^{\circ}$  to  $1^{\circ}$  lower in the morning before rising, and  $0.5^{\circ}$  higher in the evening after a heavy meal, or after a hard day's work. Distinct variations from these figures demand explanation, and if no other cause is found tuberculosis is to be considered as the possible cause.

**Fever in the Incipient Stage.**—When taken with due precautions it will be found that a subfebrile or febrile temperature is characteristic of the evolution of active phthisis even in the incipient stage, and that the absence of fever excludes active disease. The afebrile cases of phthisis mentioned by physicians are mostly the result of faulty technic in taking the temperature. Evanescent rises are overlooked. Moreover, in these cases the instability of the temperature could be determined by ordering the patient to take some exercise. An elevation of  $0.5^{\circ}$  to  $1.5^{\circ}$  in the afternoon or after some excitement, or exertion, lasting about half an hour may be observed in some persons who have no tuberculosis, as was mentioned above; with the phthisical, however, it is more lasting. It appears that a large proportion of patients with early tuberculosis have a subnormal temperature in the early morning hours, some recording as low as  $96^{\circ}$  F., before getting out of bed.

When interpreting fever in the early stages of phthisis, we should follow Darenberg's<sup>1</sup> suggestion and consider the difference between the highest and lowest temperature. Thus, a patient with a temperature of  $99.8^{\circ}$  F. at 5 P.M. has not only  $1^{\circ}$  above normal when his morning temperature is  $96.5^{\circ}$  F., but  $3.3^{\circ}$  above normal and should be considered febrile, and when prolonged for some time, it is undoubtedly of tuberculous origin, unless some other cause is found.

**Symptoms of Fever.**—These afternoon rises can also be distinguished from other rises and from physiological elevations by the concomitant symptoms which are met with in most cases of incipient phthisis. In the latter there is an acceleration of the pulse rate far

<sup>1</sup> Tuberculose Pulmonaire, Paris, 1905, p. 59.

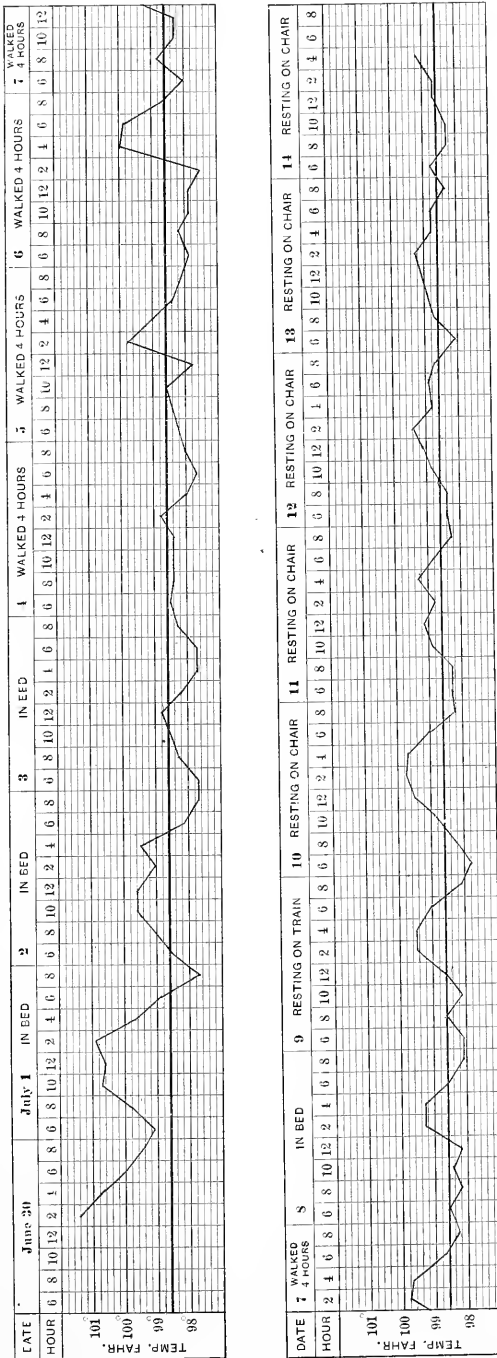


Fig. 23.—Incipient lesion at left apex, temperature taken every two hours. Showing the effects of exercise and rest on the temperature. Each time patient walked a rise in temperature occurred.

out of proportion to the slight elevation of temperature. Many also have mild chilly sensations, or even a distinct chill about an hour before the rise in temperature, when the face is pale and the extremities feel cold. Later the face becomes flushed, the eyes brighten with characteristic brilliancy, which can often be recognized by the experienced observer and the patient feels warm or hot, tired, fatigued and disinclined to work, and has some headache. It is noteworthy that despite all these symptoms the appetite for the evening meal is not diminished, which is not, as a rule, observed in fever due to other causes. Anorexia is a constant accompaniment of fever, excepting the fever of early phthisis. This tolerance of fever of the tuberculous manifests itself also in their aptitude to work during the day and sleep during the night as if they were well, feeling only somewhat tired or languid, when the thermometer reads 101° F. or more. Finally, during the night more or less sweating may occur, which even in early cases may be so profuse as to drench the patient.

**Subjective Fever without Elevation of Temperature.**—These symptoms, in varying degrees of severity, are only rarely absent in incipient phthisis and they are excellent guides in our attempts at excluding rises in temperature due to other causes. In fact, the afternoon languor just mentioned is so characteristic of the toxic state of the tuberculous that we often meet it in some advanced cases—notably, fibroid phthisis—which are afebrile. In such cases we may speak of subjective fever without elevation of temperature, first described by Dettweiler. I have seen it in a few cases of incipient tuberculosis. For this reason we must not rely solely on thermometry while treating tuberculous patients. Conversely, fever without subjective symptoms is occasionally, though very rarely, seen in incipient cases and the prognosis is very good indeed.

**Provoked Fever.**—The heat centre is apparently easily disturbed in phthisis and as a result we have usually a labile or unstable temperature. Conditions which in the average normal individual have no effect on the temperature may elevate it in the consumptive. Thus, a heavy meal, moderate exertion, emotional disturbances like reading or writing a letter, worry, anxiety and excitement, especially during the early morning hours, may raise the temperature from 1.5° to 3° F. and more. I have seen the excitement of a medical examination raise the temperature of a patient in my office 3.5° within one-half hour, and in European sanatoriums it is a routine measure to inject water at the beginning of a tuberculin treatment with a view of ascertaining whether the febrile reaction is really due to the tuberculin or to emotional disturbances. On visiting days in sanatoriums a large proportion of patients have higher fever than on other days. It has also been observed that a change in residence, as the admission into an institution, a railway journey, giving a sanatorium patient leave to spend a day with his family, etc., may elevate the temperature of the consumptive.

This *fièvre provoquée*, first described by Daremberg, and then again by Penzoldt,<sup>1</sup> can be utilized for diagnostic purposes in cases suspected of incipient phthisis. When we have a patient presenting indefinite symptoms and signs of tuberculosis but the temperature is normal, we may take the temperature before and after active exercise, and if it is raised 1° F. or more, we are probably dealing with a case of incipient tuberculosis. The usual rule is to let the patient walk about two miles and note the effect. My way has been to ask the patient to take his rectal temperature before he starts out for my office, and then walk one and a half or two miles while coming. Immediately on his arrival his temperature is again taken, preferably with the same thermometer.

A rise of 1° or more in the temperature after such a test is highly suspicious of tuberculosis; Daremberg insists that it is conclusive. Combined with other symptoms and signs, it is undoubtedly of great value. But in obese persons this may be observed without any

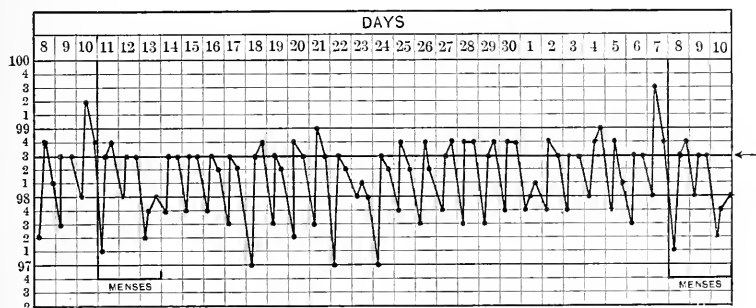


FIG. 24.—Female, aged nineteen years. Premenstrual fever in an afebrile case of incipient tuberculosis. (Bray.)

tuberculous lesions in their lungs and the same is true of anemic, especially chlorotic young women. But in physiological rises after exercise the elevated temperature again sinks to normal within half an hour of rest, while in the tuberculous it lasts much longer, two hours or even more.

**Menstrual Fever.**—In women the fever may be more accentuated during the menstrual period, which at times is of diagnostic importance (Fig. 24). We must, however, remember that in many non-tuberculous women slight elevations of temperature are observed a few days before or during that period. But in the phthisical we meet not only with elevation of temperature, but occasionally also with an increase in the number of rales over the site of the lesion, hemoptysis and pleuritic pains. Macht<sup>2</sup> says that “the rise in temperature may occur in afebrile patients, that is, patients who ordinarily run no fever as well as in those who run a slight temperature through the month.

<sup>1</sup> Handbuch der Therapie, Jena, 1910, iii, 188.

<sup>2</sup> Amer. Jour. Med. Sci., 1910, cxi, 835.

These rises may occur in early cases as well as in advanced, and in the former are of considerable diagnostic importance. If a patient shows a constantly recurring menstrual rise in temperature, and pelvic disease cannot be found, a tuberculous process should always be borne in mind."

In most cases the fever declines with the appearance of the flow; it may last several days or only a few hours. Sabourin<sup>1</sup> has shown that in certain women the menstrual fever lasts three weeks and leaves the patient only one week before the onset of the next menstruation. In these cases it is of grave importance; the patients "are killed by their courses," as Sabourin says.

Many authors, notably Vandervelde, Sabourin, Wiese,<sup>2</sup> C. A. Welch,<sup>3</sup> E. C. Morland,<sup>4</sup> and others, state that premenstrual fever indicates latent or active tuberculosis and should be given attention when attempting to make a diagnosis in doubtful cases. This premenstrual fever occurs a few days before the onset of menstruation and may continue throughout the days of the flow. Considering that it has been found that in from 40 to 50 per cent. of tuberculous women there is hyperthermia before and during that period, while in healthy women the percentage is considerable less, these authors maintain that it is of immense diagnostic value, and that the absence of menstrual fever excludes active tuberculosis.

According to Macht, these rises in temperature, when reaching high, are an evil omen prognostically; on the other hand, if they grow less, or disappear altogether, it is a sign of a cured or an arrested condition.

**Evaluation of Fever in Tuberculosis.**—In the usual case of chronic phthisis in the incipient stage there is a subfebrile temperature which is often overlooked unless the thermometer is used every two hours for a week or two. The feeling of languor which overtakes the patient during the afternoon is often taken as an indication of neurasthenia, the anorexia is attributed to dyspepsia, and the real cause overlooked. From Fig. 25 it will be seen that if in this case the temperature had been taken only at 8 A.M., 12 M., and 8 P.M., as is usually done, the febrile reaction at three to six would have been overlooked, and the patient pronounced afebrile. In rare cases, these febrile reactions occur during the night and thus escape detection. Still rarer is the so-called "reversed type" of fever, the febrile reaction occurring during the early morning hours. It appears that the prognosis is unfavorable in the last class of cases.

Since a subfebrile temperature for one or two days is no conclusive proof of the existence of active phthisis, because such ephemeral hyperthermia may be due to other causes, and also because there

<sup>1</sup> *Revue de Médecine*, 1905, xxv, 175.

<sup>2</sup> *Beitr. z. Klinik d. Tuberkulose*, 1912, xxvi, 335.

<sup>3</sup> *Lancet*, 1910, i, 639.

<sup>4</sup> *Ibid.*, 821.

are afebrile days during the incipient stage of phthisis, the temperature should be taken continuously for two or three weeks in doubtful cases before arriving at a conclusion. The readings thus plotted on the chart are the best graphic criteria for diagnosis.

The slight afternoon rises in temperature characteristic of incipient phthisis are not exclusively met with in this disease; there are other conditions which may produce hyperthermia for weeks, greatly simulating phthisis. For this reason we must not hastily decide in favor of this disease unless there are other symptoms and signs of lung disease. I have had under my care a woman who was treated for several months in a sanatorium, then handed over to surgeons for operation for gall-stones, and while convalescing after the operation another diagnosis of tuberculosis was made. The woman was then admitted under my care and for three months the afternoon temperature was almost invariably elevated  $1^{\circ}$  to  $3^{\circ}$ . We finally gave her work as a

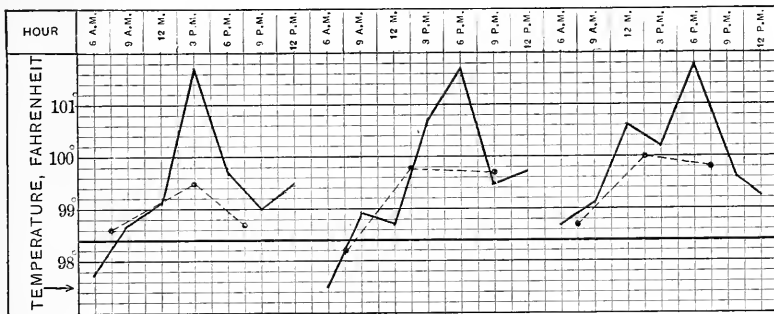


FIG. 25.—Incipient phthisis, active lesions in left apex. Temperature taken every three hours (black line) shows daily exacerbations of the fever reaching  $102^{\circ}$  F. in the afternoon. This exacerbation would be missed if temperature was only taken three times a day, at 8 A.M., 12 M., and 7 P.M., as is shown by dotted curve.

nurse and she worked during the succeeding six months quite hard and has not developed phthisis, nor shown any indications of the disease on physical exploration of the chest. She still has an elevated temperature every afternoon. These afternoon rises in temperature, when not due to tuberculosis, are mainly found in women. Anemia, especially chlorosis, and occasionally pernicious anemia may be the cause. However, an examination of the blood clears up the case. Purulent conditions of the nose and accessory sinuses, chronic inflammatory conditions of the tonsils, non-tuberculous bronchiectasis, pyelitis, diseases of the female genitalia, etc., may be accompanied by subfebrile temperature. These are but a few of the conditions which must be looked for in doubtful cases.

After all, purely hysterical fever must be borne in mind when everything else has been ruled out. There is no question but that it does occur, although our modern views of the pathogenesis of fever

are against it. This appears to be one of the many paradoxes in clinical medicine.

In evaluating the significance of the temperature range in active phthisis, we may be guided by the rules laid down by Harris and Beale:<sup>1</sup> The higher the day temperature, the more active the disease, except in a few rare instances (the so-called "reverse type") where the ordinary fluctuations are reversed, and the night temperature remains lowest throughout the whole course of the disease. But whether the normal or the inverted remissions take place, the lowest temperature is always high, and so long as it follows this course, it may be assumed that active deposition of tubercle is taking place, even though the physical signs remain for the time unaltered.

Most patients with fever lose in weight, but there are many exceptions, and patients as well as physicians are apt to judge a case more by the scale than by the thermometer. This is wrong. There are cases of phthisis, especially those in whom the fastigium occurs during the night, that remain stationary or gain in weight while the process in the lungs keeps on progressing. In other words, neither fever, nor

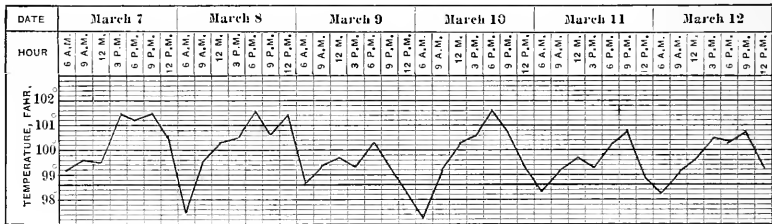


FIG. 26.—Fever in incipient tuberculosis. Temperature taken every three hours.

the weight alone should be taken as a criterion for prognosis, but all the concomitant symptoms and signs should be considered in this connection.

On the other hand, the absence of pyrexia, while a good sign in most cases, is not conclusive evidence of the mildness of the process, especially when other symptoms of active disease are present. I have seen many patients in whom the temperature never exceeded 101° F., or was even less, still the anorexia, emaciation, cough, hemoptysis, etc., were all active in bringing them to a fatal termination. This is especially seen in cases which have lasted for some years. The organism has adapted itself to the disease and does not react any more to the same degree that it does usually, and its defensive forces are in abeyance. It may be seen with any lesion, not excluding large, but usually dry, cavities in the lungs.

**Types of Fever in Chronic Tuberculosis.**—In progressive and also in advanced cases of phthisis the fever is not typical, and a diagnosis

<sup>1</sup> Treatment of Pulmonary Consumption, London, 1895, p. 314.



cannot be made from an analysis of the temperature curve alone, as is often the case in malaria, relapsing fever, typhoid, pneumonia, etc. In phthisis we may meet with any type of hyperthermia in different patients, and in the same patient at different times, depending on the activity of the process, mixed infection with pyogenic organisms, softening of lung tissue, free drainage of necrotic foci, etc. Under the circumstances we cannot speak of a typical tuberculous fever, but we meet with certain temperature curves which serve as good and reliable guides in our attempts at ascertaining the condition of the patient, the presence or absence of complications, and especially when attempting to formulate a prognosis.

**Continuous Fever.**—This is met with especially in cases with extensive pneumonic involvement, in acute pneumonic phthisis, and in tuberculous bronchopneumonia in children. In chronic phthisis which has pursued a favorable course, when a continuous temperature develops

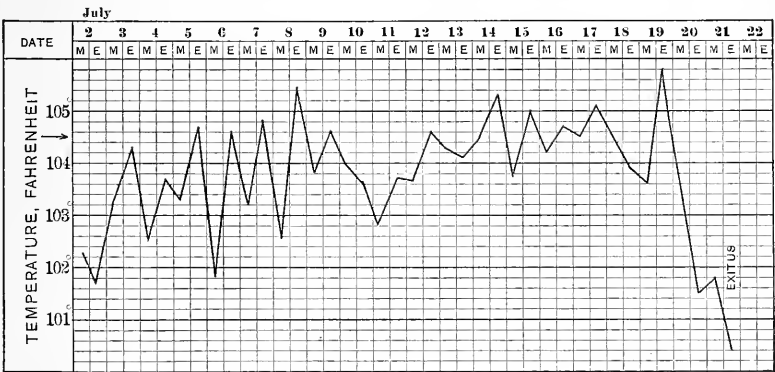


FIG. 27.—High, continuous fever in the terminal stages of pulmonary tuberculosis.

after a pulmonary hemorrhage, or without any visible cause, we may conclude that there has occurred an extension of the process in the lungs; and if this high, continuous temperature—even when it does not exceed 103° F.—lasts more than three or four weeks, the prognosis is very grave and a fatal issue may be looked for. In some cases a slight improvement may occur, but it is noteworthy that they are never cured.

**Cyclic Fever.**—In many cases of chronic phthisis we meet cyclic or undulating types of hyperthermia. The patient is never free from fever, but for two or three days during the week the maximum reading reaches 102.5° or 103.5° F., or even more, while the other four or five days it is much lower—100.5° to 101.5° F. These wave-like fluctuations may appear more or less periodically for months and not only show variations during each week, but the febrile waves may appear at greater intervals, every two or three or four weeks, as can

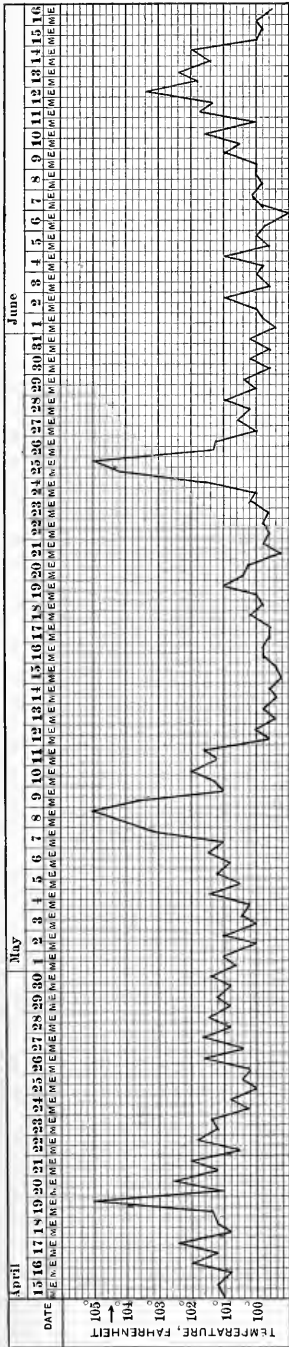


Fig. 28.—Active tuberculosis with continuous fever, marked by acute exacerbations, occurring at irregular intervals.

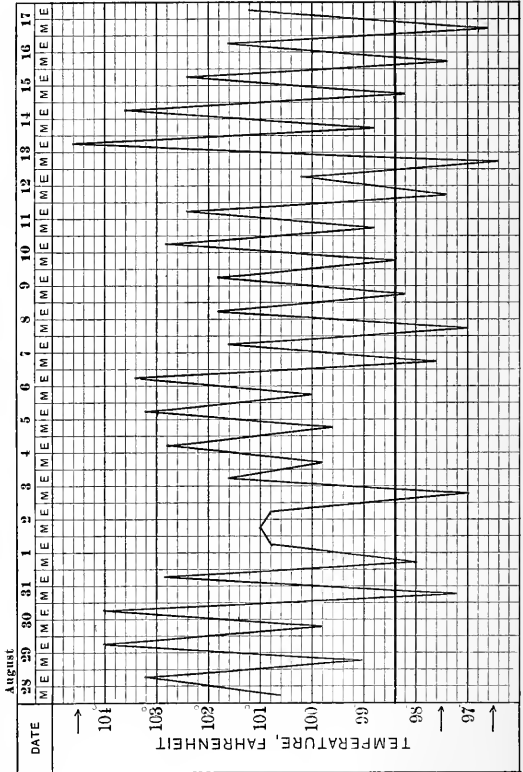


Fig. 29.—Hectic fever in advanced cavitory phthisis.

be seen from Fig. 28. It is seen in cases in which old foci are softening or the pulmonary process is extending and each exacerbation of the fever is an expression of a new area of involvement which may in many cases be easily discerned by a careful physical exploration of the chest.

**Hectic Fever.**—In progressive disease these types of hyperthermia are usually followed at the end by hectic fever (Fig. 29). In cases in which there is softening in the lung, the necrotic tissue being gradually expelled leaving cavities, the temperature chart tells the story. There are morning remissions during which the temperature is nearly normal or even subnormal, while in the afternoon there may be a chilly sensation, or a distinct chill with chattering of the teeth; the pulse which was rapid and small during the apyrexial morning hours, is even more accelerated, the temperature begins to rise reaching  $103^{\circ}$ , and in some cases even  $105^{\circ}$  at about five in the afternoon. The night sweats in these cases are very profuse and exhausting.

The time of the highest fever in these hectic cases is variable. Often the maximum is attained in the afternoon, but in many it is around noon and in the evening it may be normal. If in such cases it is taken only mornings and evenings, we may find a record of normal temperature, because the midday rise, which may have been quite high, has been overlooked.

This hectic fever may last for weeks, or even for months, during which time the unfortunate patient is reduced to a skeleton by the fever and the accompanying anorexia and diarrhea, which are hardly ever lacking. The frightful appearance of the bundle of bones with hardly any visible muscles, which have atrophied extremely, covered by a clammy, muddy skin; the skin emaciated but edematous around the ankles and knees, the eyes deeply set in the orbits, the temples sunken, is disheartening to the physician making his rounds in the hospital; he feels helpless when the slowly sinking, but still struggling, human being gazes appealingly for assistance which cannot be given. It is noteworthy that with all this material decay the intelligence and often the hopes and aspirations of the patient are well retained, and he begs for the relief of some minor and comparatively insignificant symptom such as the cough or diarrhea, saying that if this is removed he will feel in excellent condition.

At the terminal stages there may be irregular fever, the curve of one day differs from that of the other. Saugman<sup>1</sup> states that this is a good sign of intestinal tuberculosis when occurring in the earlier stages of the disease (Fig. 30).

**Subnormal Temperature.**—The subnormal temperature seen in many incipient cases during the morning hours has already been mentioned. But we also meet with patients in the advanced stages of the disease

<sup>1</sup> In Brauer, Schröder, and Blumenfeld's *Handbuch der Tuberkulose*, ii, 284.

who present subnormal temperature throughout the day and night for weeks; the mercury never rises above 98.5° F., and early in the day it may be as low as 96° or 97° F. The disease may be active and even progressive, yet the thermometer gives no indication of it. I have many of these cases in my hospital service. I find it is usually an indication of excavation, just as fever is an indication of infiltration, caseation, and softening of lung tissues.

These cases have been recently spoken of by O. K. Stone:<sup>1</sup> "At certain periods of the disease, usually succeeding the active febrile stage, there is often a period when the temperature curve shows marked excursions in the subnormal, the temperature at no time rising above 98.6° and rarely fully reaching this point. The patients during this period of subnormal temperature are usually improving and making distinct gains, but it takes very little to give them exacerbations of real temperature, lasting for a few hours to a few days."

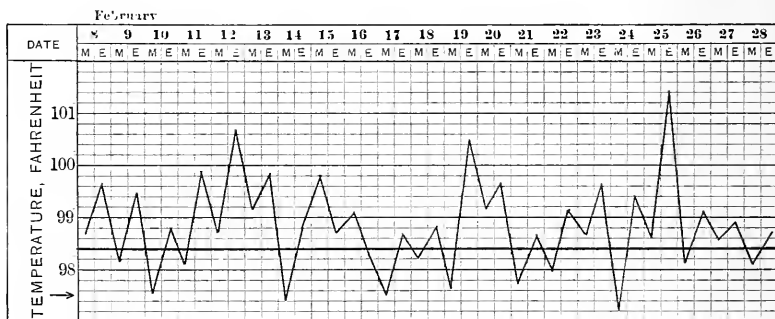


FIG. 30.—Irregular fever in advanced tuberculosis of the lungs with intestinal complications.

Subnormal temperature is also seen in fibroid phthisis and emphysema complicated by tuberculosis, in both of which the disease runs a chronic, sluggish course. Many keep disabled for years, though not confined to bed, but they never fully recover. A subnormal temperature is also seen on rare occasions in a subacute case of phthisis which suddenly took a turn for the better after the necrotic tissue in the lung had been eliminated from that organ and a cavity remained. In this class recovery may take place, as I have seen on several occasions.

The sudden drop in the temperature combined with dyspnea and cyanosis in a febrile case of phthisis may mean a spontaneous pneumothorax, or a rapid extension of the necrotic process in the lung overwhelming the patient. The prognosis in either event is grave indeed. In many extremely emaciated consumptives the temperature is subnormal for several days before death.

<sup>1</sup> Boston Med. and Surg. Jour., 1914, clxxi, 1008.

**Apyretic Tuberculosis.**—In old chronic cases of phthisis we may have a normal temperature for months, though the process in the lungs keeps up. This is seen in fibroid phthisis, in phthisis in the aged, and in tuberculous pleurisy. Many of these patients live for years and do not lose in weight. I have seen such patients last for fifteen and twenty years, always ailing, coughing, expectorating, at times having spells of more or less profuse hemoptysis. They are the important sources of the dissemination of tubercle bacilli; more so than most of the stormy cases. They are not strong enough for muscular work but may be moderately efficient at any occupation which does not require undue exertion. We meet these cases mainly among the well-to-do, who can afford to lead an idle life, or among the very poor who have intrenched themselves in hospitals for chronic and "incurable" cases of tuberculosis and, for one reason or another, like institutional life, and stick to it for long periods. We also meet these active but apyretic cases among the more cultured classes, who either know how to take care of themselves or, being professional persons, they may pursue their vocations with more or less efficiency. Some are very brilliant, and the type of consumptive drawn by so many writers of fiction is usually copied after the model of this class of patients. It is noteworthy that while most of them are more or less emaciated, we now and then meet one who is actually fat and may even be placed in the category of the obese. They usually suffer from dyspnea because of the fatty heart and pulmonary fibrosis.

Phthisis in the aged also runs an apyretic course at times and, because they do not cough excessively, the disease may not be recognized.

It appears that there are great differences in the reactive powers of different persons suffering from phthisis. In some the fact that they have a normal temperature is no proof that the disease is benign, especially if other symptoms of active disease are present. I have seen patients whose temperatures hardly ever exceeded 101° F., yet they wasted, perspired, and had exhausting diarrhea; they finally died with a low temperature. While the temperature curve is an excellent guide as to the tendencies and progress of the disease, these apyretic cases must be judged more by the general symptoms than by the thermometrical findings, as has already been shown.

**Fever due to Complications.**—During the course of phthisis fluctuations in the temperature usually go hand-in-hand with the activity of the disease, and each elevation or depression in the temperature curve may be explained by the findings in the chest through physical exploration. But there are exceptions. Many elevations of the temperature are due to non-tuberculous complications. Thus, as will be seen from Fig. 31, malaria may complicate phthisis and produce confusion, unless the blood is examined and the malarial parasite is found.

Other complications to be mentioned are constipation, acute gastritis, tonsillitis, influenza, pleural effusions, etc. These may be the

cause of a sudden elevation of temperature in a case in which the tuberculous process is proceeding rather favorably. Careful examination usually reveals the cause of the pyrexia.

A rise in the temperature in a tuberculous patient may be due to the administration of certain drugs, mostly of the sedative and hypnotic class, as has been pointed out by Sabourin<sup>1</sup> and Mantoux.<sup>2</sup> I have repeatedly observed that after the administration of opium or its derivatives, morphin, codein, heroin, dionin, etc., or chloral, sulfonal, trional, etc., there is often a rise in the temperature during the succeeding twenty-four hours. A rise of this kind is especially vivid when occurring in an afebrile patient to whom one of these drugs has been administered. The fever lasts no more than twenty-four hours,

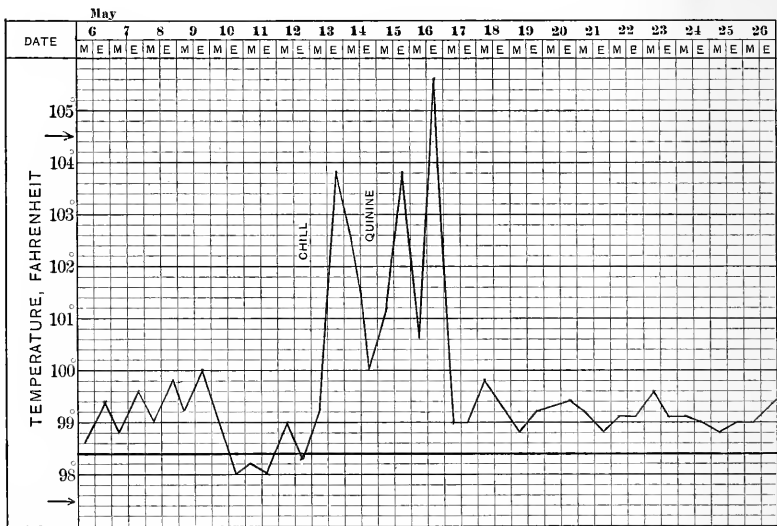


FIG. 31.—Malaria complicating phthisis.

as a rule, but I have seen cases in which it lasted longer. Hypodermic medication is more apt to act this way, and Mantoux says that injections of salt solution may also elevate the temperature.

**Diagnostic and Prognostic Significance of Fever in Phthisis.**—Summarizing the results obtained in this section, we may say that in a patient who shows a distinct elevation of temperature during the afternoon for several weeks, and no other cause can be found, tuberculosis is to be thought of. If it is provoked by moderate exercise, and persists after more than an hour of rest, it is almost pathognomonic of phthisis. If with it there are other symptoms, such as nightsweats, anemia, loss of weight, cough, emaciation, etc., tuberculosis is in all probability

<sup>1</sup> Rev. gén. de clin. et de thérap., 1906, xx, 639.

<sup>2</sup> Revue de la tuberculose, 1907, iv, 395.

the cause, even if the physical signs are not definite. The diagnosis is more certain if the morning temperature is subnormal.

In the course of the disease a high temperature during the day, never touching the normal, and ascending in the evening is an indication of progressive activity of the process in the lung. The disease is progressing slowly, or is even quiescent, when the temperature in the early morning on rising is subnormal or normal and remains so during the day, not rising above 101° F. late in the afternoon or evening.

High, continuous temperature, above 103° F., is an indication of extension or dissemination of the disease in the lung, and if it lasts for more than a month, a fatal issue is to be expected; even if some improvement is noted, recovery should not be expected.

Hectic fever, with normal or subnormal temperature in the morning and high fever, 103° or more at midday or later is an ill omen. While the patient may keep on in this condition for weeks or months, he will in all probability never leave his bed alive.

In most cases, absence of fever is an indication of an improvement or a cure of the disease, but there are many exceptions, and the other constitutional symptoms must be considered when formulating a prognosis. A subnormal temperature, when coming on suddenly, is a bad sign. When chronic, lasting for several weeks, however, it is not incompatible with an inactive, though not necessarily an inefficient, life.

### NIGHTSWEATS.

Nightsweats have at all times been considered pathognomonic of phthisis. A prolonged cough will not alarm the average person, but when it is associated with nightsweats, he will soon consult a physician with a view of ascertaining whether or not he is tuberculous. They are met with quite early in the disease in many cases; at times when the characteristic symptoms and physical signs are lacking, but in advanced cases their severity does not depend altogether on the extent of the lesion.

**Causes.**—The causes of nightsweats are obscure. Some have attributed this phenomenon to the compensatory activity of the skin when the pulmonary respiratory area is diminished, but we meet them in cases with but little damage to the lung. Gustav Heim<sup>1</sup> is of the opinion that the products of cell disintegration, and especially the toxins produced by the bacilli, stimulate the sweat centre directly or reflexly. Just as after childbirth the remains of the placenta may produce sweating. It is an attempt on the part of the body to rid itself of harmful matter, just as it is excreting carbon dioxide in the sweat when this is excessive in the blood. Smith and Brehmer have attributed the nightsweats to the quick change of the tachycardia of the day to the bradycardia of the night. Because stimulating food, like

<sup>1</sup> Zeitschr. f. Tuberkulose, 1910, xvi, 365.

milk punches, often prevent nightsweats, they find therein a confirmation of their theory.

It appears that Cornet's theory is more in harmony with the facts observed clinically. He looks upon nightsweats as due to the absorption of the proteins of the tubercle bacilli and other microorganisms secondarily implanted in phthisical lesions. The toxins are absorbed into the blood stream and they stimulate the heat centre, thus causing fever; and also act upon the sweat centre in the cord and medulla and the peripheral secretory glands and thus produce perspiration. He shows that this also confirms the fact that in spite of the great disturbance the diminished excretion of fluid and the greater difficulty in the elimination of carbon dioxide which is characteristic of the chronic course of the disease as compared with acute phthisis, the secretion of sweat is incomparably less in the former, owing solely to the more gradual absorption of the toxins.

**Symptomatology.**—Nightsweats usually occur in the second part of the night, about 2 to 4 A.M., in typical cases. The patient retires with some fever, and in hectic cases may have had a chill on the preceding afternoon, sleeps rather restlessly, is disturbed by dreams or by cough, and wakes up during the early morning hours drenched with perspiration. At times, changing the night- and bedclothes may prevent their recurrence during the same night, but in many cases this is of no avail, as the sweats again trouble the unfortunate victim.

In the milder forms, the sweating may be local, on the forehead, the neck, the chest, etc. Rarely it is noted on only one side of the body, usually the one corresponding to the pulmonary lesion.

In the progressive and hectic cases the sweating may be so profuse and drenching as to exhaust the patient who often begs for the relief of this symptom alone which, together with the diarrhea, is instrumental in relieving him permanently from his earthly sufferings.

It is important to mention that the nightsweats do not directly harm the patient, considering that only 1 per cent. of solids is eliminated in this way, of which 0.7 per cent. is salts, mainly uric acid. Only so far as disturbing sleep is concerned are nightsweats harmful. In children their diagnostic significance is less than in adults. (See Chapter XXIV).

In some cases the disease runs its course without any or only with slight nightsweats. Kuthy found that 37 per cent. of his patients had nightsweats during the first stage of the disease. In the third stage, 61.5 per cent. According to this author, women are more apt to sweat profusely than men. But Louis found only 10 per cent. of cases without nightsweats, and at the Phipps Institute they were absent in 41 per cent. of 3344 cases.

In the evolution of phthisis it is observed that the sweats run hand-in-hand with the fever and the general condition of the patient. During afebrile periods they are absent, to return with an acute exacerbation. There are said to have been found cases of nightsweats without



fever, but my experience leads me to believe that the fever is overlooked in such cases. One of the best signs of improvement is the complete disappearance of the nightsweats.

Nightsweats may be prevented in a large proportion of cases by the adoption of hygienic bedding and coverings during sleep, as will be shown in another part of this book.

Sweating appears to be easily provoked in the phthisical. Kuthy and Wolff-Eisner say that not only consumptives, but also those "predisposed" sweat easily and when waking find themselves bathed more or less in perspiration. Mild exertion, grief, worry, excitement, etc., may be followed by more or less profuse perspiration, general or local. In a large proportion of patients we see sweating in the armpits during medical examination, even in patients who do not sweat during the night. We also meet with patients who sweat during the day while taking a nap, etc.

While most authors, notably Cornet, state that the sweat does not carry infection, recent investigations by Piéry have shown that it may contain bacilli which are pathogenic to animals. Salters showed that hypodermic injections of the sweat into animals act like tuberculin.

## CHAPTER X.

### HEMOPTYSIS.

**Frequency.**—To the layman the most reliable symptom of pulmonary tuberculosis is blood-spitting and many physicians share this view, although we know that a large proportion of cases of phthisis pursue their course and terminate in recovery, or fatally, without any hemoptysis, while in many patients hemoptysis is not due to tuberculosis. The statistics of the frequency of this symptom vary considerably, some finding it in but 25 per cent., while others report as many as 80 per cent. having had hemorrhages during the course of phthisis. Sokolowski says that advanced consumptives who did not bleed from time to time are only rarely met with. Louis found this symptom in 65 per cent. of cases; Walshe<sup>1</sup> in 80 per cent.; Wilson Fox<sup>2</sup> says that more than one-half of all cases of phthisis present this symptom in some part of their course; Williams found it in 70 per cent.; Sargo<sup>3</sup> in 38 per cent., Condie in only 24 per cent., and Elmer H. Funk in 54 per cent.

These wide differences in the percentages are easily explained by the fact that the authors have not taken their figures from comparable material. Some have spoken only of fatal cases, other of cases in their private practice, while still others have taken hospital records as their criteria. In the latter classes the patients were observed only for a short time, and hemorrhages which may have taken place later have not been considered.

Anders<sup>4</sup> found in a series of 5302 cases that 36.6 per cent. had hemoptysis. He emphasizes, however, that not all were followed until the death or recovery of the patients, but many were discharged during the course of the affection. In fact, among 289 cases in private practice, kept under observation for a longer time, as a rule, hemoptysis occurred in 41.8 per cent., but it is to be recollected that even these patients were under observation for less than half of their duration. Hemorrhage is more apt to occur in advanced cases, and those who base their calculations on early cases in sanatoriums are likely to find low percentages, while when only fatal cases are taken the percentages will be too high.

**Initial Hemoptysis.**—Of great interest is hemoptysis as an initial symptom of phthisis. But statistics on this subject are also at variance, because we meet with many patients who have been coughing and

<sup>1</sup> British and Foreign Med. Chir. Review, 1849.

<sup>2</sup> Dis. of the Lungs and Pleura, London, 1891, p. 785.

<sup>3</sup> Brauer, Schröder, and Blumenfeld's Handbuch d. Tuberkulose, ii, 250.

<sup>4</sup> Jour. Amer. Med. Assn., 1907, xlix, 1067; 1909, liii, 455.

presented other symptoms of tuberculosis for months, or even years, and paid little attention to them till a hemorrhage brought them to their senses. Here it would not be correct to consider the hemoptysis as the first symptom.

In a study of 1932 cases Reiche<sup>1</sup> found that 9.2 per cent. had more or less profuse hemorrhage at the beginning of the disease, and in one-fourth of these it was rather copious. He finds that those who bleed at the beginning are more apt to bleed during the course of the disease than those who do not; the ratio is 57.9 per cent. and 31.7 per cent. Sorgo found during a period of observation extending over ten years that 12.9 per cent. of 5872 patients had initial hemorrhages. Kuthy<sup>2</sup> reports that while 54.3 per cent. of his patients had hemoptyses, only two-fifths of these (22.3 per cent.) were initial hemorrhages. Anders arrives at the conclusion that in about 10 per cent. of cases of phthisis, hemoptysis first directed attention to, and is almost invariably followed by, demonstrable and conclusive evidence of the disease; but in not less than 25 per cent. of all cases of chronic pulmonary tuberculosis, hemoptysis is among the ushering-in symptoms of the active recognizable period of the affection.

**Pathology.**—The diagnostic and prognostic significance of hemoptysis can only be appreciated when we have a clear understanding of the anatomical changes responsible for the bleeding. There are several varieties of pulmonary lesions which may bring about exudation of blood from the lung tissues: Local inflammatory or active hyperemia; ulceration of a bloodvessel, and aneurismal dilation of bloodvessels are the most important in phthisis.

The initial hemoptyses are said to be caused merely by localized, active inflammatory hyperemia. In other words, they are of the same origin as the rusty sputum of pneumonia. But we may well understand that this bleeding, caused by diapedesis, cannot be profuse—only blood-streaked sputum may thus be brought about. This is met with in all stages of phthisis and can only be accounted for in this manner. On the other hand, blood-streaked sputum does not invariably mean that it is caused by localized hyperemia and that the lesion is not serious, because not all the extravasated blood is brought out through the mouth. Quite some of it remains in the lungs and bronchi, and is more or less quickly absorbed, as was shown by Nothnagel. When the hemorrhage is not profuse we must not conclude that the case is mild or that the lesion is not extensive.

When the pulmonary lesion proceeds from infiltration to caseation, then to softening and finally to liquefaction, it undoubtedly implicates the bloodvessels that pass through it and produces the same changes in the lung tissue which surrounds it. It is therefore strange at first sight that, considering the ulcerative processes and the destruction of tissue, hemorrhages do not occur more often. But this is explained

<sup>1</sup> Zeitschr. f. Tuberkulose, 1902, iii, 223.

<sup>2</sup> Die Prognosenstellung bei der Lungentuberkulose, p. 299.

by the strong tendency to the formation of thrombi in the blood-vessels, excepting in very acute cases. In chronic cases there usually occurs a narrowing or complete obliteration of the vessel by the growing tubercles which, when finally ulcerating, may leave an erosion through which the blood can flow more or less freely till it is occluded by a thrombus. Moreover, the increased blood-pressure at the infected and inflamed area dilates the softened vessels and causes small aneurisms which have been described elsewhere. This is clear when we bear in mind that the bloodvessels in the lungs are terminal branches of the pulmonary artery. These aneurisms may easily rupture and permit blood to escape.

Most cases of hemoptysis end in recovery, and the pathological changes in the lung at the time of the bleeding can only be surmised, but in fatal hemorrhages we often have an opportunity to observe the anatomical changes. Here we usually find that the source of the bleeding was an exposed vessel, left bare after the surrounding pulmonary tissue had softened and was eliminated. The loss of support, as well as the pathological changes in the perivascular tissues, and the erosions of the tunica adventitia and media, lead to aneurismal dilations of the inner coat which give way to the pressure exerted on them by the circulating blood.

The rupture of these aneurisms at times strikes down a patient who is on the road to recovery when a hemorrhage occurs like a storm out of a clear sky. When the cavity into which the aneurism or the lacerated artery opens is small, the extravasated blood usually coagulates and the clot obstructs the opening of the bloodvessel, thus stopping the bleeding. But in large cavities, or when the blood is deficient in coagulability, which is not rare, the bleeding keeps on till the patient dies of acute anemia. I have seen at autopsy a large cavity filled with about a quart of blood which killed a patient during the night. After clearing out the clots we found an eroded artery about 2 mm. in diameter, and passing a probe through it, we found it only about 6 cm. from the pulmonary artery. This patient had such a sharp hemorrhage that he was unable to call for assistance.

In more acute cases of phthisis, in which the destruction of lung tissue is going on at a rapid pace, the hemorrhages usually come from ulcerating erosions of large pulmonary vessels and may prove fatal immediately. Here there is no time for narrowing of the blood-vessel, thus preparing it that in case of rupture it may be easily repaired by occlusion with a thrombus which saves the majority of chronic consumptives from death due to this cause. In acute pneumonic phthisis which very often begins with sharp and profuse hemorrhage, I have usually been able to find signs of cavitation when the acute process subsided and the disease pursued a chronic or subacute course. This confirms the view that profuse hemorrhage is not caused by mere active inflammatory hyperemia, but by actual erosion of a bloodvessel.

In fibroid phthisis the sources of hemorrhages are lacerated, dilated or varicose bloodvessels which pass through bronchiectatic cavities, characteristic of this form of the disease and also oozing from capillaries or arteries which traverse the granulations on the walls of the cavities. The bleeding is therefore not profuse, as a rule, in these cases, but it is recurring in many cases.

**Hemoptysis at the Onset of Phthisis.**—As the first symptom to draw the attention of the patient to his affection, hemoptysis occurs in two different types. We meet it in patients who have felt perfectly well till the instant the hemorrhage made its appearance without any premonitory symptoms. Even close questioning does not elicit any symptoms preceding the bleeding. While at work, or engaged in an animated conversation, or even waking up from sleep during the night, the patient feels a sensation of warmth in the throat, coughs and expectorates a mouthful of blood; or during a fit of coughing he brings up some blood-streaked sputum. A careful examination of the chest and skiagraphy may fail to disclose anything conclusive of pulmonary disease. The temperature is and remains normal, the appetite is good, but for a few hours or days the patient continues to bring up dark clots, and when this ceases he is apparently none the worse for his experience. Many of these patients subsequently pass through life without experiencing anything that may lead to the suspicion of tuberculosis. This is seen in many who have passed through an attack of abortive tuberculosis, details of which are given later on. Some patients give a history of such a hemorrhage many years before the onset of active phthisis.

In many others the initial hemorrhage continues for several days and when it finally ceases the patient shows symptoms of phthisis—cough, expectoration, tachycardia, nightsweats, etc. Physical exploration of the chest reveals distinct signs of a lesion in one or both apices and tubercle bacilli may be found in the sputum. The subsequent course of the disease is that of chronic phthisis, though a large proportion of cases are aborted within a few months, and I have met with patients who have had several attacks of hemoptysis at long intervals, have shown some indefinite or even conclusive apical signs, and rarely tubercle bacilli in the sputum, yet they remained well indefinitely.

A different clinical type of hemoptysis is seen in patients who maintain that they have felt quite well, but close questioning reveals the fact that they have been coughing for months, bringing up mucopurulent sputum; that the appetite has failed and that they have lost weight and strength. In women we may find that they have missed one or more of their periods. They, however, considered these symptoms trifling and continued at their work, or consulting a physician, they were told that it was only a slight "cold."

The hemoptysis in these cases is apt to be profuse and last for several days because, while insidious in its arrival, the tuberculous process

in the lungs has usually progressed quite far; indeed I have met with signs of pulmonary excavations in such "initial" hemorrhages. In the majority of cases physical exploration of the chest reveals a lesion of moderate extent, though on rare occasions we find nothing definite, even with the aid of skiagraphy. But the cough, fever, nightsweats, expectoration, etc., continue and the diagnosis is made without conclusive physical signs. In most cases tubercle bacilli are found in the sputum. It is the prolonged and slow convalescence after the attack of hemoptysis that distinguishes these cases from the initial hemorrhages of abortive tuberculosis.

**Hemorrhages during the Advanced Stages.**—In confirmed chronic cases of phthisis we may meet with hemoptysis at any period of the disease, though it may be added that it is most frequent in the early and very late stages. The bleeding may be of various degrees, from that of sputum tinged with blood to the expectoration of several mouthfuls of pure, bright red blood, to a copious hemorrhage during which several pints are brought up within twenty-four hours, and in rare cases it has been reported that as much as three quarts of blood were brought up.

The blood is bright red, frothy, usually mixed with sputum. When bleeding is very profuse the blood may be "blue" or venous. It is evident that in most cases the blood does not coagulate quickly—some clots are seen, but the bulk remains fluid; even the addition of calcium salts, serum and tissue extracts does not enhance its coagulability. E. Magnus Alsleben<sup>1</sup> has added normal blood without increasing its power of coagulation. The reasons for this delayed coagulability are not clear.

Many patients have some premonitory warning before the onset of hemoptysis, and I have had one who could foretell bleeding twenty-four hours in advance. At times there is a rise in temperature, and pains in the chest are aggravated, or the cough becomes more annoying. But in most patients the onset is sudden and unexpected. The patient has a sensation of gurgling or tightness in the chest, followed by a fit of cough productive of bright red, frothy blood which has a salty taste and partly coagulates in the vessel into which it is deposited, forming flattened lumps. When very profuse, which is comparatively rare, the patient is overwhelmed and can hardly cough—the blood gushes in an almost steady stream through the mouth and at times through the nose.

The general appearance of the average patient is that of shock—he is prostrated, often out of proportion to the amount of blood lost; his countenance is that of a frightened individual, unnerved, anxious and terrified; the face pale, the extremities cold and clammy. The temperature, which may have been above normal before the onset of the bleeding, suddenly sinks, often to a subnormal degree, the pulse is rapid, soft and small.

<sup>1</sup> *Zeitschr. f. klin. Med.*, 1914, lxxxix, 9.

That these symptoms of collapse are not due wholly to the loss of blood is evident from the fact that the family is also panic stricken, and some are in the same state of collapse as the patient, showing the profound influence this symptom has on the average person.

After getting some reassuring encouragement from his physician there is usually observed a reaction in the patient—the pulse improves, the face becomes flushed and the temperature rises to the same degree as it was before the onset of the bleeding, or higher. In many cases there is soon a relapse, the bleeding is repeated within a few hours or the next day and it may keep on at irregular intervals for a week or more. When it finally stops the patient continues to expectorate dark blood-clots with his sputum for several days. In some cases the bleeding continued for weeks, letting up for a day or two to reappear; rarely until the patient expires from exsanguination, cerebral anemia, and cardiac asthenia.

In cases with large pulmonary cavities the bleeding may be very copious. The quantity of blood brought out is not all that has escaped from the bleeding vessel. A considerable part is swallowed automatically, and some remains in the cavities or the bronchi and is subsequently absorbed. The outcome of the bleeding depends on the size of the cavity and the coagulability of the blood. In rare cases the weak and emaciated patient is overwhelmed by the bleeding and is unable to expel it from the lungs, expiring in a few minutes, drowned or suffocated by his own blood. Other patients make a vain fight for hours or days but finally succumb to exsanguination. But the chances of recovery of a bleeding patient with a cavity in the lung are, on the whole, not bad. An immediate fatal issue is, after all, exceptional; less than 2 per cent. of consumptives die from hemorrhage directly. The vast majority of hemorrhages are well borne, the patient dying, if at all, from other symptoms or complications.

On the other hand, we meet with patients who have made an excellent recovery, but suddenly profuse hemorrhages occur which carry them off within a few hours or days. I was once called to attend a patient who was discharged from a sanatorium three days previously as an arrested case of phthisis. He succumbed to the bleeding. These hemorrhages are fortunately rare and are usually due to the rupture of an aneurism in a dried and contracted cavity. They can neither be foreseen nor prevented.

**Hemorrhages in Fibroid Phthisis.**—In this form of phthisis hemoptysis is very frequent. In most cases it is very slight, only blood-tinged sputum being brought up. They may feel quite well in general, excepting for the dyspnea and the cough to which they have adapted themselves. But no sooner does blood make its appearance in the sputum than they are alarmed. I have, however, had some patients who did not mind the blood-tinged sputum much, knowing from experience that it is not at all dangerous.

**Hemorrhagic Phthisis.**—There is a form of phthisis which is characterized by frequent and recurrent hemorrhages, the *hemorrhagic phthisis* of the old writers. The bleeding occurs at irregular intervals for years without harming the patient very much. In these patients we may not find any definite physical signs in the chest, no fever, no pronounced emaciation, and but little cough. Only the hemoptysis and, at times, the bacilli in the sputum reveal the condition. I have had under my care at the Montefiore Home a woman in whom neither any of the other physicians nor myself were quick in making a diagnosis of tuberculosis from the indefinite physical signs and the skiagram of the chest. In fact, we had suspected malingering and employed strong measures to make sure that the temperature readings were not influenced by manipulations of the thermometer, and that the sputum was expectorated by the patient, suspecting that there was some deception on the part of the patient, who liked to remain in the hospital. Even during the more or less copious attacks of hemorrhage which recurred at frequent but irregular intervals and often lasted for several weeks, no conclusive physical signs could be elicited in the chest. I have another patient who has bled at least twice a year for the past fifteen years and feels quite well. Andral mentions a case which bled off and on for sixty years and finally succumbed at the age of eighty to some disease of the chest. These cases are uncommon but we meet them now and then. In some we find signs of more or less extensive pulmonary lesions which remain stationary or quiescent in spite of the recurring hemorrhages. The lesion is benign notwithstanding the tubercle bacilli which are found in the sputum, and at times, though rarely, there may be one hemorrhage which proves fatal. It has been stated that in most of these cases the lesion is localized in the tracheobronchial glands.

**Exciting Causes of Hemoptysis.**—We have seen that while hemoptysis is rather common among consumptives, still many pass through the disease till the end, recovery or death, without this accident. There appears to be some evidence showing that tall persons are more likely to bleed than those of shorter stature, and Wolff states that for this reason women show a lesser proportion of bleeders than men. Strandgaard<sup>1</sup> suggests that the tall patients are more likely to bleed because they have larger hearts and higher blood-pressure, but this view has not been confirmed. While hemoptysis has been seen at all ages, even in infants, still most of the cases occur between fifteen and fifty, probably because at this period most of the cases of phthisis are active.

From Ander's statistics it appears that males are more liable to hemoptysis than females and prior to the twentieth year of age there is a slight preponderance in favor of the female sex. In Thompson's<sup>2</sup> collective investigation the women showed greater liability than the

<sup>1</sup> Zeitschr. f. Tuberkulose, 1908, xii, 209.

<sup>2</sup> Causes and Results of Pulmonary Hemorrhage, London, 1879.



men. But Anders shows that this increased incidence in the female sex is confined principally to the first two decades of life. After the thirtieth year the number of males preponderates. Females are also less liable to suffer from copious and fatal hemorrhages. My own experience coincides with that of Anders, that an immediately fatal hemorrhage is relatively rare in women. Initial hemoptysis is also less frequent in women than in men. Reiche's statistics show that it occurred in 11 per cent. of the latter as against only 5.5 per cent. in the former; Sörgo found the ratio as 11 and 13.5 per cent. respectively; while Berthold Müller<sup>1</sup> found it in equal proportion in both sexes.

Patients with a nervous and excitable temperament are more apt to suffer from this complication than the indolent and phlegmatic. During some animated conversation, overexertion, singing, running, mountain climbing, straining at stool, or as a result of traumatism, hemorrhage may be provoked. It is also a fact worthy of the careful attention of clinicians that consumptives who have been urged on to eat excessively, becoming plethoric, ruddy and fat, bleed more often than those who eat well, but moderately. Exposure to the inclemencies of the weather may excite hemoptysis, probably by causing an acute localized pneumonic process at the site of the tuberculous lesion. Coitus may excite it and I have known two cases of fatal hemorrhage which occurred soon after intercourse.

Certain drugs used extensively in phthisiotherapy, as arsenic, creosote and its derivatives, the iodides, aspirin, etc., are often instrumental in bringing on hemoptysis. It has been stated that residence in high altitudes favors hemoptysis, but it has not been proved; as will be shown elsewhere, the prognosis of hemorrhage is worse in these regions than at sea level.

Some authors have found that there are seasonal influences in the production of hemoptysis, saying that the spring and summer months give the highest incidence, while Anders's collective investigations show that it is most prevalent in the months of December, January, and February; August, September, May and March, in the order named, seem to rank next. The experience at the Phipps Institute coincides with those of Anders. Burns<sup>2</sup> says that "barometer changes seem to have little effect on the symptomatology. In a few instances hemorrhages have occurred following a fall in the barometer but in insufficient number of cases to justify constant relation. It is probably a matter of coincidence only so far as the barometer alone is concerned. There is a larger number of patients streaking in March, May and especially June than in other months. Hemorrhage occurred more frequently in June than in any other month."

I have observed in my hospital work that hemorrhages often occur in epidemic form, a large number of patients bleed at the same time in a ward. This may be explained by some intercurrent infection,

<sup>1</sup> Zeitschr. f. Tuberkulose, 1910, xiii, 133.

<sup>2</sup> Boston Med. and Surg. Jour., 1914, clxx, 564.

especially influenza, causing pulmonary congestion. But psychic influences may also be at work.

Any of the above-mentioned factors may be the apparent exciting cause, but this is not true of the majority of cases. In my experience, a large proportion of hemorrhages, especially copious ones, begin when the patients have the least reason to expect them. It is the universal experience in sanatoriums that patients who have been kept under a rigorous rest cure may bleed. Furthermore, about one-half the hemorrhages begin during the night when the patient is resting in bed or sleeping, and suddenly wakes up with a cough, followed by a mouthful of blood. In patients with eroded bloodvessels or miliary aneurisms in the lungs, bleeding is apt to occur without any known provocative cause, and usually it cannot be prevented by any known means.

**Diagnostic Significance of Hemoptysis.**—It has been repeatedly stated that all cases of hemoptysis should be considered of tuberculous origin and treated accordingly until proved to be due to some other cause. But just because the vast majority of hemoptyses are due to tuberculosis of the lungs, when the blood is derived from some other source, it at times proves a serious source of error. I have seen hematemesis treated as hemoptysis on several occasions. Though this could be easily avoided by carefully noting the manner in which the blood is brought out, yet I have seen two cases in which this was not so simple and an immediate diagnosis could not be made.

The most perplexing cases that present themselves in physicians' offices are patients who claim that several days ago they expectorated blood. In many the blood was derived from the nose, throat, gums, etc. Examination of these parts may not reveal any irritation, hyperemia or varices, while in the chest there are found some indefinite signs of an apical lesion which may be of non-tuberculous origin, thus leading to an erroneous diagnosis of tuberculosis. This is especially seen in cases of epistaxis in which the blood trickled down the posterior nares, exciting cough productive of blood or blood-streaked sputum. Some patients have epistaxis during the night, wake up spitting blood and present themselves promptly in the morning for a medical examination which does not reveal any definite clues as to the source of the bleeding.

**Streaky Sputum.**—Great care must be exercised before diagnosing tuberculosis based on a history of blood-streaked sputum. While this, when originating in the lungs, may be a precursor of a large and profuse hemorrhage, it is, however, a fact that streaky sputum only rarely originates in the pulmonary parenchyma; the vast majority comes from the nose, throat and especially the bronchi. West<sup>1</sup> says that streaky hemoptysis is far more frequent in bronchitis than in phthisis. When it occurs in phthisis it is generally due to the same cause, viz., the rupture of distended capillaries in the bronchial tubes

<sup>1</sup> Diseases of the Organs of Respiration, London, 1909, ii, 381.

as the result of violent coughing; but when the tubes are the seat of tubercular ulceration, bleeding may sometimes take place from the ulcerated surface, usually in small amount and streaky, but occasionally in larger amount.

In many cases with a history of streaky sputum the diagnosis can only be cleared up by careful observation for weeks, after the presence or absence of fever, tachycardia, anorexia and physical signs in the chest are carefully studied. Very often the blood is derived from congestion in chronic pharyngitis with a spongy mucous membrane, or from dilated or varicose bloodvessels in the trachea or main bronchi common in asthma and chronic bronchitis. Varicosities of the esophagus are also said to be quite common. Recently Gorel and Gignoux<sup>1</sup> have described *fausses hémoptyses* due to varices at the base of the tongue which are visible in the laryngeal mirror. The vein may be large and dilated and often extends to the fold of the epiglottis, or only a number of blue or dark blue specks may be noted, at times confluent, greatly resembling a vascular tumor. These are very often causes of hemoptysis. They are found mostly in persons between forty and fifty years of age, especially those who show other stigmata of arteriosclerosis and other varicosities, as on the legs, or hemorrhoids.

These false hemoptyses have been described by many English physicians. Williams<sup>2</sup> speaks of persons who, without any symptoms of lung disease, bring up quantities of blood and recover without permanent cough. He says that they were generally middle-aged and often had the arcus senilis. Recovery is the rule. Sir Andrew Clark<sup>3</sup> also describes "arthritic hemoptysis" occurring in elderly persons free from ordinary disease of the heart and lungs; a form of hemoptysis arising out of minute structural alterations in the terminal bloodvessels of the lung. These vascular changes occur in persons of the arthritic diathesis, resemble the vascular alterations found in osteo-arthritic articulations, and are themselves of an arthritic nature. More recently F. de Havilland Hall<sup>4</sup> attributed these hemorrhages to high vascular tension. Even though it occurs in a patient who has had phthisis, this form of hemoptysis is not necessarily due to a recrudescence of the disease, but may be the result of high tension with degenerate vessels.

**Hemoptysis in Heart Disease.**—Blood-spitting in heart disease is often treated as of tuberculous origin with disastrous results. Inasmuch as we very often meet with cardiacs who are emaciated, cough and have occasionally mild pyrexia, the diagnosis of tuberculosis is at times made erroneously. It is in fact usually supported by some physical signs in the chest, because cardiacs may show defective

<sup>1</sup> Lyon Méd., 1911, xliii, 1913.

<sup>2</sup> Pulmonary Consumption, London, 1887, p. 135.

<sup>3</sup> Trans. Med. Society of London, 1889, xii, 9; Lancet, 1889, ii, 840.

<sup>4</sup> Lancet, 1915, ii, 329.

resonance, alteration in breath-sounds and even rales over an apex, or other parts of the chest as a result of infarction, peripheral thrombosis, or brown induration. I have seen cases of organic heart disease treated in tuberculosis clinics and day camps in New York City for months. In infarction the expectorated blood may be bright red, but in mitral disease small, solid, purple or black lumps which sink in water are usually brought up. They are derived from ruptured capillaries in the walls of air cells where they remain for some time before they are expectorated. The experienced eye can generally distinguish them.

According to Frederick W. Price,<sup>1</sup> mitral stenosis is probably the next most frequent cause of hemoptysis to pulmonary tuberculosis and a common cause of mistake. Perhaps the heart is not examined at all, or if it be examined it is by no means rare for the characteristic murmur to be absent. Furthermore, because there are frequently apical signs, as has already been indicated, phthisis is often diagnosed. In several cases I was nearly trapped by this similarity of mitral disease to phthisis, but noting some irregularity in the heart beat, I investigated further and diagnosed mitral stenosis. It must always be remembered that while active phthisis is not altogether excluded with heart disease, yet it is extremely rare, especially in mitral stenosis.

**Hemoptysis in Bronchiectasis and Syphilis of the Lungs.**—In bronchiectasis bleeding is not uncommon, and I have seen copious hemorrhages due to this cause. The blood is derived either from dilated and congested bloodvessels in the proliferated mucous membrane, or from inflammatory changes in the mucosa, or from small corroded aneurisms in the walls of bronchiectatic cavities, similar to those found in tuberculous excavations. As a rule it is encountered in older persons. During the hemorrhage the diagnosis may be difficult, though a careful history clears up the case. In syphilis of the lungs, hemoptysis of various degrees has been encountered.

**Menstrual Hemoptysis.**—Phthisical women, if they are to have hemoptysis at all, are more apt to have it during the menstrual period. It has been observed that during menstruation there is usually an increased blood-pressure and a congestion of the laryngeal mucous membrane, and some state that active periodical hyperemia of the lungs occurs at that time and this would favor extravasation of blood, especially in the affected area. According to Macht<sup>2</sup> these periodical hemorrhages, which may be very slight or profuse, may persist after the patient has improved in health and the tuberculous process becomes arrested. Periodic hemorrhages in consumptives at the time of menstruation may take place from other organs than the lungs. Thus, Wilson and Newman have reported such hemorrhages from the trachea and upper respiratory passages. Macht also reports a rather

<sup>1</sup> Brit. Med. Jour., 1912, i, 287.

<sup>2</sup> Amer. Jour. Med. Sci., 1910, cxl, 835.

interesting case of a woman with pulmonary tuberculosis with intestinal complications—ulcer in the bowels—who regularly had severe hemorrhages from her intestines at her periods.

Vicarious menstruation, which is very rare, appears to be due in most cases to tuberculosis. But in evaluating vicarious menstruation it must be borne in mind that amenorrhea is very frequent in phthisis, and in this disease hemoptysis is frequent; it is therefore not surprising that hemoptysis should occasionally occur while the menstrual flow has been delayed or suppressed.

**Localization of the Source of the Hemorrhage.**—Heretofore the determination of the side of the chest in which the bleeding takes place was merely of academic interest because it made very little difference on which side the ice-bag, which has been traditionally used in the treatment of this symptom, was applied. But recently, since we found that an artificial pneumothorax may stop a copious hemorrhage after everything else has failed, it is of practical importance to localize the bleeding-point.

In cases which have been under observation for some time and it is known that the lesion is unilateral, the problem may be simple, inasmuch as profuse bleeding implies an old cavitory lesion. But in bilateral cases it is difficult, often impossible, to determine positively which lung is bleeding. Percussion must not be done for fear of increasing the bleeding and auscultation may be of service in showing a limited area of moist, consonating rales, and perhaps amphoric breath-sounds. But it is a noteworthy fact, which must never be lost sight of, that during profuse hemorrhages the blood may be aspirated into the non-bleeding lung and produce all sorts of rales. It is therefore, at times, impossible to decide positively which lung is bleeding.

In rare cases we hear murmurs, synchronous with the heart beat, over the site of excavations. Gerhardt found that these murmurs originate in arteries which traverse the walls of cavities and he verified his observations at the autopsy table. In several cases this phenomenon was observed by me, the murmur was audible below the clavicle, and over the same area were most of the classical signs of pulmonary excavation. These patients are apt to bleed copiously, and they often succumb to a sharp hemorrhage. Here we know that the source of the bleeding is the branch of the pulmonary artery which traverses the cavity, and operative treatment (an artificial pneumothorax) may be attempted when a hemorrhage cannot be controlled otherwise. But these cases are rare and in the average case we cannot say with any degree of certainty that the bleeding vessel is located in a superficially recognized excavation, and not in another one, either located deeper, or altogether in the other half of the chest. I have repeatedly seen cases in which after a copious hemorrhage the more affected side remained unaltered, while in the unaffected lung new rales appeared.

According to Stricker,<sup>1</sup> the bleeding comes from an eroded vessel when it occurs suddenly during the course of acute and progressive phthisis, while in chronic cavitory phthisis it is usually derived from an aneurismal dilatation of a vessel. Repeated hemorrhages accompanied by fever point to progressive decay of the affected area in the lung. Hemoptysis in the advanced stages of phthisis is derived from eroded arteries, and for this reason the prognosis is less favorable than in hemoptysis in incipient cases or in initial hemorrhages, which are, as a rule, of venous origin.

**Differential Diagnosis.**—In cases of initial hemoptysis it is imperative to ascertain whether the blood is derived from a tuberculous lesion or is due to some other cause. Careful examination of the nose and throat may reveal that it is altogether due to congestion or varicosity of these mucous membranes, as has already been mentioned. When the sanguinous fluid expectorated is uniformly bright red and watery, it is in all probabilities derived from the mouth. In case no symptoms or signs of a pulmonary lesion are discovered, and the bleeding cannot be ascribed to a non-tuberculous condition, the heart is normal and there is no history of an injury, the patient is to be placed under prolonged observation before deciding that he is not tuberculous. But it must always be borne in mind that mere streaks in the sputum may be due to many causes other than tuberculosis of the lungs, and a diagnosis of phthisis should not be made because of their presence alone.

In copious hemorrhage, when it is not feasible to examine the patient's chest carefully, it is often difficult to decide whether the bleeding is due to a tuberculous lesion, a bronchiectatic cavity, pulmonary syphilis or, in rare cases, whether it is not altogether hematemesis. The last-mentioned condition may simulate hemoptysis because the patient may have aspirated the blood into the respiratory passages and then expectorated it; while in hemoptysis the blood may be swallowed and then vomited. It may then greatly simulate blood derived from the stomach, viz., black or chocolate-colored, thick lumps or clots, mixed with the contents of the stomach. I have met with cases in which the diagnosis could not be made immediately. We may, however, be guided by the following points: In hemoptysis the blood is, as a rule, coughed up, bright red, frothy and mixed with sputum. It is also alkaline and does not clot. Auscultation may reveal rales in some part of the chest, and a careful history will show that the patient has been coughing, expectorating, etc., for a long time, while in cases of hematemesis the history points to disturbances in the gastric functions, and physical signs may be discovered in the abdomen. In hemoptysis we invariably observe that after the cessation of active bleeding the patient keeps on coughing and expectorating clotted blood for several days, which is never observed in hematemesis.

<sup>1</sup> Nothnagel's Handbuch d. spez. Pathol., xiv, 7.

But when the hemorrhage from either source is brisk and copious, and there is no history, the points just enumerated are often of no value, because the blood is bright red, alkaline, and not mixed with either sputum or gastric contents. But such profuse hemorrhages are only seen in advanced consumptives and there are always to be noted the stigmata of tuberculosis.

In cases in which the diagnosis has not been previously established, bleeding from the deeper respiratory passages may, on rare occasions, be difficult of differentiation as to whether it is derived from a tuberculous lesion or from a bronchiectatic cavity. I have been guided by the pulse and temperature of the patient—when these are normal, and the general condition of the patient is good, the chances are that there is a bronchiectatic cavity, especially in persons over forty-five years of age. In older persons with arteriosclerosis the so-called “arthritic diathesis” is to be thought of. Usually a careful history clears up the diagnosis, while in rare borderline cases we should reserve our opinion till the hemorrhage ceases and a careful examination of the patient is feasible.

In addition to tuberculosis the following conditions are liable to cause pulmonary hemorrhage: Cardiac disease, hemophilia, bronchiectasis, syphilis, abscess and gangrene of the lung, certain acute specific fevers, pneumonia, suppurative processes in the mediastinum, foreign bodies in the bronchi, injuries to the chest, paroxysms of pertussis, echinococcus, cancer, actinomycosis, aspergilosis, hydatid, distoma pulmonum, and pneumokoniosis.

**Prognostic Significance of Hemoptysis.**—Patients, almost without exception, overestimate the significance of hemoptysis and are more terrified at the appearance of a speck of blood in their sputum than by any other symptom or complication of phthisis, excepting perhaps spontaneous pneumothorax. It is for this reason that initial hemoptysis has been described by some authors as a salutary phenomenon, because it draws the attention of the patient to the condition of his lungs which he may have otherwise neglected. In fact, I have known cases in which hemoptysis was actually life-saving for just this reason in patients who had coughed for months and presented other symptoms of phthisis which they considered a trifling affair, when, like the climax of a slowly developing drama, hemoptysis made its appearance, opening their eyes, or even those of their physicians, so that proper treatment was instituted.

A hemorrhage may prove fatal immediately or within a few days of its appearance; or, if the patient survives it, it may have an influence on the course of the disease.

**Prognosis in Initial Tuberculous Hemoptysis.**—We have already mentioned that many cases of pulmonary hemorrhage, even when due to tuberculous lesions, are not necessarily followed by symptoms of phthisis. Every physician has among his clientele patients who have coughed out more or less blood years ago and have never suffered

from disease of the lungs. "Outspoken tuberculosis does not necessarily follow hemoptysis," says Frederick T. Lord,<sup>1</sup> "which may occur in patients with apparent good health and sound lungs. Of 329 instances of hemoptysis observed by Ware, 62 (18 per cent.) recovered without subsequent symptoms to suggest pulmonary tuberculosis. The interval elapsing between the attack of hemoptysis and the last report was over ten years in 41 cases. In 1768 Goethe, at the age of nineteen years, and then a student at Leipzig, had an attack as follows: 'One night I waked with a severe hemoptysis and had enough strength and presence of mind to wake my room-mate . . . for several days I wavered between life and death.' For some months he thought he had pulmonary tuberculosis and must die young. At the age of eighty-two years he had hemoptysis again and died at the age of eighty-three years. His long and active life may serve as a comforting example to those who need encouragement. At the age of twenty-three or twenty-four years, Rousseau expectorated blood and gave up his work as a teacher of singing. He died at the age of sixty-six."

**Proportion of Deaths due to Pulmonary Hemorrhages.**—When profuse, the patient may be exsanguinated and succumb to cerebral anemia, or the blood may overflow the bronchial tree and suffocate him, especially when it occurs suddenly while the patient is asleep. While this outcome is seen now and then, it is a very rare occurrence. Louis had but 3 fatal cases in 300 consumptives; Williams 4 out of 198 fatal cases; Wilson Fox 4 out of 101; Moeller saw only 1 fatal hemoptysis during fifteen years' experience with consumptives; Wolff reports a lethal outcome three times among 1200 tuberculous patients (0.25 per cent.); Winsch 1 among 200 (0.5 per cent.); Thue, 13 times among 975 patients (1.6 per cent.); Sargo 14 deaths among 5800 consumptives (2.4 per cent.) and among 2.16 per cent. of his patients subject to hemoptysis. Lord reports that death as an immediate result of bleeding occurred in only 1 of 76 patients with hemoptysis at the Channing Home, and 2 of 142 at the Massachusetts General Hospital. Death as a consequence of extension of pulmonary infection for which the hemorrhage was responsible, occurred in 1 case at the Channing Home, and 6 other cases at the Massachusetts General Hospital.

Williams reports that in 1000 cases, including 63 fatal ones, where the patients had hemoptyses of one ounce and upward on one or more occasions, the average duration was seven years and six months; an average differing only by a few months from that of the total deaths. In 200 living cases of similarly extensive hemoptysis, the average was eight years and three months—about the same as that of the living cases generally. "It is only in the far-advanced stages that it is likely to curtail the duration of the disease. In early cases hemop-

<sup>1</sup> Diseases of the Bronchi, Lungs, and Pleura, Philadelphia, 1915, p. 360.



tysis is comparatively unimportant," concludes Williams. When we say that hardly one out of a thousand deaths due to tuberculosis is caused by hemorrhage, we are as near the true figure as possible.

**Influence of Hemoptysis on the Course of Phtthisis.**—The influence of hemorrhage on the course of the disease is misunderstood by the average patient and often overestimated by the physician. It may be said that as long as it does not prove fatal immediately, and this is rare as we have just shown, it has no effect on the patient nor on the disease. Many older writers have stated that it often has a rather salutary effect, and not altogether without reason, as is proved by the course of many cases subsequent to hemorrhages. Lebert, Flint, Wilson Fox and others state that hemorrhages may produce a sense of relief, and cough and expectoration previously existing may temporarily disappear. Williams says, "To many patients its occurrence seems beneficial rather than otherwise, for the congestion is thus relieved and the system not materially weakened by the loss of blood." I have seen many cases in whom the disease took a turn for the better soon after a more or less profuse hemorrhage, and others in which the cough, anorexia, pains in the chest, etc., disappeared after this accident. We know that slight abstraction of blood is often beneficial inasmuch as it stimulates the blood-forming organs to produce more blood cells.

The fear, formerly entertained, that the blood, spreading all over the bronchial tree, is apt to inoculate new areas and produce new lesions in hitherto unaffected parts of the lung, is now known to be without sound foundation because reinfection is difficult or even impossible in the vast majority of cases. To be sure, we find that the bronchi contain blood while auscultating a patient during, or immediately after, a hemorrhage, but this is usually transitory, disappearing by absorption or expectoration within a few days after the bleeding ceases and the original pulmonary lesion, if not progressive, remains the same as it was before, pursuing the same course as if no such accident had occurred. Cases in which after a hemorrhage a quiescent lesion begins to pursue an acute or subacute course, and tuberculous bronchopneumonia is found at the autopsy, are, in all probabilities, due to a sudden reduction in the powers of resistance, about the causes of which we know nothing at present. They do occur now and then, but when taken in connection with the large number of hemoptyses in which this sequel does not occur, they are comparatively rare.

More than sixteen hundred years ago Galen stated that the prognosis of pulmonary hemorrhage depends on the fever which is apt to accompany it—afebrile cases recover, while in febrile cases the prognosis is gloomy. More extended experience in recent years has confirmed the opinion of this ancient and empirical clinician.

In hemoptysis the immediate and especially the ultimate prognosis depends less on the bleeding, its abundance or even repetition, than on the extent of the pulmonary lesion and the symptoms which accompany or dominate the clinical picture, the subsequent course

of the original disease—phthisis—and the complications which may arise. When we find during a hemorrhage that a patient has a good, full pulse, less than 100 in frequency, and no fever or dyspnea, the immediate prognosis is good. If there are several repetitions of the hemorrhage during the subsequent few days, the prognosis is, as a rule, favorable as long as the pulse is good and there is no fever. Even fever is of no grave significance if it lasts but a couple of days. It is then due to absorption of the blood remaining in the bronchi. It is only when the fever is high and persistent for several days that it assumes serious significance.

In case the pulse becomes small, soft, compressible and rapid we may be sure that the bleeding continues even if we do not see it brought up in large quantities through the mouth, for we may have internal hemorrhage in phthisis, the blood being retained in a large cavity, while the feeble patient is unable to force it out by cough. This is especially apt to occur after large doses of morphine have been administered, or in severely emaciated persons.

In cases which had been active before the onset of the bleeding, having had fever, tachycardia, emaciation, etc., the prognosis after cessation of the bleeding is usually the same as it would have been had there been no such complication. The temperature usually drops during a brisk hemorrhage, but it rises again and the course of the disease continues unabated. But if the temperature has been normal, or only slightly above, and the pulse is less than 100, full and bounding, the patient has a good appetite, and sedative drugs are judiciously, if at all, administered, the immediate as well as the ultimate outlook is indeed good.

In most cases the findings on physical exploration of the chest after moderate hemoptysis remain the same as they were before that event, although on auscultation we usually hear moist, consonating rales which may not have been there before the onset of bleeding. These rales may persist for several weeks. In some cases we find that the area of dulness over the upper lobe extends because of caseous or necrotic changes engendered during the hemorrhage. This dulness may disappear after the clots have been absorbed, or after the resolution of the pneumonic areas. More frequently it is in time supplanted by tympany due to excavation.

## CHAPTER XI.

### SYMPTOMS CAUSED BY DISTURBANCES IN THE GASTRO-INTESTINAL TRACT—THE SKIN— THE JOINTS.

#### GASTRO-INTESTINAL SYMPTOMS.

**Frequency.**—Some authors have stated that phthisis develops mostly in individuals who have been naturally bad eaters; others have maintained that those suffering from gastric derangement are most likely to fall prey to the disease, and Grancher says that “all consumptives have been, are, or will become, dyspeptics.” In practice we meet with many patients who have been treated for gastritis for a long time until the true nature of their disturbance became evident. The diagnostic and especially the prognostic significance of anorexia or gastritis in a disease which depends in its origin and outlook on proper nutrition cannot be overestimated.

As far back as 1826 Wilson Philip<sup>1</sup> drew attention to the fact that many cases of phthisis are preceded for some time by severe indigestion. In his excellent monograph on the “Dyspepsia of Phthisis,” W. Soltau Fenwick<sup>2</sup> quotes Todd, Sir James Clark, Budd, Bennett, Ansell and other writers of the first half of the nineteenth century to the effect that dyspepsia is a very frequent forerunner of phthisis. In those days some authors even spoke of “gastric phthisis,” and “pretuberculous dyspepsia” is even now mentioned by some authors. There is no doubt that incipient phthisis, as we know it at present, was in those days not recognized, and this was responsible for the notion that phthisis is often preceded by dyspepsia.

Recent investigations, however, do not confirm that gastro-intestinal disturbances are *per se* predisposing factors in the evolution of phthisis, though Fenwick says that for his own part he is quite convinced that there does exist a variety of dyspepsia which is peculiarly apt to be followed by pulmonary tuberculosis.

As an early symptom of phthisis, dyspepsia is quite frequent. Hutchinson<sup>3</sup> found it in 92 per cent. of his cases, and in 55 per cent. it was quite severe. Samuel Fenwick, Dobell, Pollock, and others have found it in nearly similar proportions. W. Soltau Fenwick states that “dyspeptic phenomena of sufficient severity to attract the attention of the patient are encountered in about 70 per cent. of all cases of early phthisis, but that the early development of the disorder in any individual case depends to a great extent upon the sex of the

<sup>1</sup> Treatise on Indigestion, London, 1826, p. 323.

<sup>2</sup> The Dyspepsia of Phthisis, London, 1894.

<sup>3</sup> Medical Times, 1855, i, 583.

patient, the type of the tubercular disease and the previous condition of the digestive organs." He found that it is more apt to occur in females than in males and in general in that variety of phthisis which commences insidiously and progresses slowly.

More recent investigations have only partly confirmed the findings of the above-mentioned clinicians, and there are writers who consider anorexia, though not a result of gastritis, a constant symptom of incipient phthisis, like fever, cough, nightsweats, emaciation, etc. An analysis of 3007 cases in the Phipps Institute showed that 55.3 per cent. presented symptoms referable to the stomach. It appears, however, as H. R. M. Landis<sup>1</sup> shows, that these gastric disturbances were in no way due to changes in the stomach peculiar to tuberculosis itself; the changes being such as might occur in any chronic wasting disease. Janowski<sup>2</sup> reports that among 700 patients, 35 per cent. suffered from gastric disturbances, which were more often encountered in women than in men. With this Kuthy is also in agreement. He found that in 37.3 per cent. of his male patients there were gastric disturbances as against 50.1 per cent. in his female patients. In the first stage, 38 per cent.; in the second stage, 46.4 per cent.; and in the third stage 57.2 per cent. showed these symptoms.

**Symptomatology.**—One of the characteristics of the anorexia of phthisis is that, unlike the appetite in other diseases, it is independent of the fever in many cases. Many patients with but slight fever have an almost complete antipathy for food, while others who have moderate fever, preserve an excellent appetite. Laségue said "all patients who eat and digest their food well despite having fever are consumptives." In acute pneumonic phthisis, which is often difficult to differentiate from lobar or lobular pneumonia, I have placed great reliance on this symptom: In pneumonia the anorexia is invariably complete, while in acute phthisis the appetite may be retained more or less, and in spite of a temperature of 103° or 104° F. the patient is apt to ask for nourishment.

In incipient phthisis the appetite is often very capricious. One day a certain food is preferred while the next it is despised and morbid cravings are not uncommon, especially in women. A large proportion of patients cannot tolerate certain kinds of food—some will not eat meat, others refuse milk, eggs, etc. It seems to me, however, that the repugnance for milk and eggs is often not the result of the tuberculous process, but is an acquired characteristic due to the stuffing with these articles of food which is so commonly carried to an extreme degree. Following the usual advice "plenty of milk and eggs" is likely to ruin an excellent appetite if carried to extremes. Two or three quarts of milk and half or one dozen of raw eggs daily, which tuberculous patients often consume, may result in a strong repugnance to these articles.

<sup>1</sup> Trans. Nat. Assn. Study and Prev. Tuber., 1910, vi, 193.

<sup>2</sup> Zeitschr. f. Tuberkulose, 1907, x, 493.

An aversion to fats of any kind is very frequently observed in phthisical patients. Hutchinson noted this fact over sixty years ago and stated that 71 per cent. of his phthisical patients disliked fats; 33 per cent. could take them in but small quantities; while only 5 per cent. liked them. Fenwick noted a marked aversion to fat in 64 per cent., and many of his patients developed this peculiar antipathy many months or even years before the onset of the pulmonary disease. He observed that among families which exhibit a marked predisposition to tuberculosis, it is not uncommon to find that several members possess a strong aversion to all forms of fat and are often unable to partake of even a small quantity of this material without suffering from acidity, nausea or attacks of biliousness. Occasionally we meet with tuberculous patients who dislike carbohydrate and especially saccharine foods, the ingestion of which causes more or less severe gastric discomfort.

In many cases the anorexia improves with the improvement in the local condition in the lung; but we also meet with cases in which the tuberculous lesion is slowly progressing or quiescent, but the appetite improves, as if the organism had adapted itself to the tuberculous toxemia. In fact almost insatiable hunger may be seen on rare occasions.

In the early stages of phthisis digestion is fair or good in most cases. Indeed, it appears to me that digestion in phthisis usually depends on the condition of the gastro-intestinal tract before the onset of the lung disease. As was already intimated, the excessive quantities of milk and raw eggs may be responsible for the symptoms of dyspepsia in many cases, such as pyrosis, belching, flatulence, bad taste in the mouth, etc. The fact that these symptoms may be removed by appropriate corrections in the diet is in favor of our contention. Excepting in advanced cases and in alcoholics, vomiting, if it occurs at this stage, is due to cough, as has already been detailed when speaking of the emetic cough. It is also likely to be preceded by nausea, which is not the fact with the emetic cough.

**Causes of Anorexia.**—It appears that the anorexia of phthisis is of toxic origin. Analyses of the gastric contents have not revealed any constant changes in the anatomy or functional activity of the stomach in the early stages of phthisis. In some cases hyperchlorhydria is found, in others hypochlorhydria, while in many others the free and combined acids remain in about normal proportions. Nor have any constant secretory or motor disturbances been observed. The physiology and pathology of the stomach in early phthisis, as studied by Klemperer, Hayem, Einhorn, Brieger, Fenwick and others show no characteristic functional changes.

Many French authors, notably Marfan,<sup>1</sup> are of the opinion that the gastric symptoms in early phthisis are due to the general anemia

<sup>1</sup> *Troubles et lésions gastriques dans la phthisis pulmonaire*, Paris, 1887.

which causes sluggish secretion of gastric juice, weakness of the smooth musculature and hyperesthesia of the gastric nerve endings of the vagus. Fenwick, finding that the dyspepsia in phthisis is not a direct result of pyrexia, nor of direct irritation of the mucous membrane, concludes that it is probably due to the chronic absorption of certain toxic substances which are manufactured in the pulmonary cavities; but he describes a form of dyspepsia which often precedes the development of pulmonary tuberculosis, when cavities are out of the question.

The gastric symptoms appear to be analogous with those observed in chlorosis and the severe anemias which cause ischemia of the digestive tract. But as Janowski points out, many tuberculous patients without any anemia also suffer from gastric symptoms, and he concludes therefore that the anorexia is not invariably due to general anemia, but to ischemia of the gastric and intestinal mucosa. This explains why so many different results have been obtained from analyses of the gastric contents. It is the paroxysmal proclivity of the gastric disturbances which is characteristic of early phthisis.

**Gastric Symptoms in Advanced Phthisis.**—The anorexia and other gastric symptoms of early phthisis usually subside in cases pursuing a favorable course and the patients recover. But in cases with progressive disease, especially those characterized by pulmonary excavations, more or less severe symptoms of dyspepsia are present. Nearly a century ago Louis found that about two-thirds of his phthisical patients had shown signs of dilatation of the stomach. W. Soltau Fenwick found among 100 autopsies in cases of tuberculosis in which he took special notes on this point, that the lower margin of the viscus extended below the level of the navel in 64 and he says that it is rare while performing an autopsy on a phthisical subject to fail to encounter some increase in the dimensions of this viscus. The degree of gastrectasis appears to bear a direct relation to the extent and chronicity of the pulmonary lesion.

Chronic catarrh is very frequent but true tuberculous ulcers are exceedingly rare, probably because the stomach contains very little lymphoid tissue and bacilli cannot reach there through this channel, and the acid secretions are inimical to the growth of tubercle bacilli. Fenwick, after a careful search was able to discover the records of 24 cases of this affection, several of which are, however, open to suspicion; while among the notes of 2000 necropsies on cases of phthisis performed at the Brompton Hospital he could find only two instances in which tuberculous ulcers of the stomach were discovered. Lauritz found 4 cases of undoubted tuberculous ulcers in the stomach among 580; Melchior 6 in 848 autopsies, and Gassmann 6.13 per cent. in 600 autopsies. There have been reported cases of perforation of tuberculous gastric ulcers into the peritoneum, though this is exceedingly rare because of the inflammatory adhesions which usually form around the ulcers and the peritoneum. Simple gastric

ulcers are not infrequently found at autopsies on tuberculous bodies, but the proportion is not higher than among patients who succumbed to any cause.

In the vast majority of cases of advanced phthisis the appetite is poor; those who do attempt to eat usually display various distastes for certain foods, and even this is not constant—the appetite is often very capricious, and many develop morbid cravings. This is one of the difficulties of feeding phthisical patients in sanatoriums and hospitals. At times we meet with patients who retain an excellent appetite to the end and cases of bulimia are not unknown. Pain after eating, pyrosis, belching, etc., are very common and vomiting is at times a prominent symptom. But while the emetic cough may be encountered in advanced cases, the vomiting at this stage is usually not of this type. They simply vomit because of gastritis, or dilatation of the stomach. This type of vomiting is usually preceded by nausea, belching, etc., and not by cough as in the other type. The nausea and retching may persist for several hours after the vomiting and the ejecta consist of sour food mixed with mucus. I have met with cases in which no food could be retained owing to vomiting and some even with hematemesis. The prognosis in these cases is gloomy indeed.

In hectic cases the gastritis is often very troublesome and, combined with vomiting, nightsweats, cough, diarrhea, etc., it is one of the terminal symptoms of phthisis. In many cases, however, the pulmonary symptoms overshadow the gastric phenomena, but very often the latter are sufficiently pronounced to require great care and attention. The amyloid liver often contributes considerably to the digestive disturbances, but lardaceous changes in the blood-vessels of the stomach are not unknown. I have met with cases of this type, extremely emaciated, hardly able to move a limb, yet they asked for food which, when given by the nurse, was relished with an apparently voracious appetite.

It appears that the dyspepsia of advanced phthisis is usually associated with pulmonary excavation, and is mainly caused by the prolonged intoxication characteristic of progressive and advanced disease. A fruitful source of gastric derangement is swallowed sputum, more common in women. The sputum not only irritates the mucous membrane of the gastro-intestinal tract, but it is also absorbed and produces toxemia. The mucous membrane of the gastro-intestinal tract eliminates poisons from the blood, which in their turn irritate these membranes, as is the case in acute mercurial poisoning in which mercurial albuminates circulating in the blood are eliminated into the intestines where they cause severe diarrhea. The injection of large doses of tuberculin may also cause diarrhea.

**Intestinal Symptoms.**—During the incipient stage of phthisis the bowels are unaffected in most cases, though we meet with constipation in a large proportion of cases. But I doubt whether the proportion is higher than among people with modern habits of life and dietetic

conditions. In some cases the constipation is due to the sedative medication used for the control of the cough.

Diarrhea may be one of the symptoms of incipient tuberculosis. It is met with mainly in patients at the two extremes of life—in children under ten years of age and in senile patients. In children the diarrhea may be the only symptom, while examination of the chest may show nothing conclusive, or signs of tracheobronchial adenopathy may be found. In aged patients who have felt quite well, even claiming that they have not coughed, a chronic and persistent diarrhea should be considered a sign that a careful examination of the chest is urgent. It will be found that there are signs of old phthisical lesions in the lungs, and the sputum may contain numerous tubercle bacilli. Very rarely diarrhea is one of the symptoms of incipient phthisis in young adults.

In some patients the functions of the bowels remain more or less normal through the course of the disease, but this is rare. In most cases diarrhea makes its appearance with the advance of the disease. While in many cases it is due to tuberculous ulceration of the bowels, there are others in which it is caused by intestinal catarrh very frequently the result of dietetic errors. In many the ingestion of large quantities of milk is responsible and eliminating milk from the diet promptly gives relief. In others the excessive amount of fat, mainly eggs, is responsible. Persons who have had intestinal trouble before the onset of phthisis are more liable to suffer from catarrhal diarrhea than others. As will be pointed out later when speaking of tuberculous ulceration of the intestine, the differential diagnosis is exceedingly difficult. The prognosis depends on the causation of the diarrhea. When due to amyloid degeneration or tuberculous ulceration of the intestines the prognosis is grave.

### EMACIATION.

Emaciation is a cardinal symptom of phthisis; one of the triad mentioned by Richard Morton, the others being cough and fever. Popular lore, as well as medical experience, have always associated tuberculosis with emaciation. Phthisis (Greek, *φθίσις*), consumption, has its equivalent in every European language. That it is mainly due to the tuberculous toxemia engendered by the metabolism of the tubercle bacilli is evident from the fact that experimental tuberculosis is always accompanied by emaciation of the animals.

In acute galloping consumption and in miliary tuberculosis the emaciation is progressive and frightful, much more rapid than in other febrile diseases, as pneumonia, typhoid, etc., and this is one of the most important points in the differentiation of acute tuberculosis from other acute diseases. In children, when during or after an attack of measles, pertussis, etc., the wasting becomes very marked



and there is dyspnea, rapid pulse, etc., acute tuberculosis is to be suspected.

While the denutrition and wasting in phthisis is often caused and always enhanced to a certain extent by the gastro-intestinal disturbances which are concomitants of the disease in all its stages, we meet with emaciation almost constantly in active disease with fair gastro-intestinal functions. Some authors are inclined to attribute the emaciation to the lowered powers of absorption caused by a congenital narrowing of the lymph channels in the intestinal tract which is said to predispose to phthisis. But this has not been proved.

**Extent of Emaciation.**—Not only is the subcutaneous adipose tissue wasting, but the nitrogen-containing muscles also vanish with astonishing rapidity. It is noteworthy that the first muscles to waste are those of the thorax—the pectorales, the scapular, the intercostals, etc. In many incipient cases we see a striking contrast between the wasted and flabby muscles of the chest—and in women occasionally the wasted breasts—and the fairly preserved contour of the muscles on the extremities. Moreover, the muscles and subcutaneous tissue of the affected side of the chest waste earlier than those on the opposite and unaffected side. The result is that the supraclavicular and suprascapular fossæ are more or less deeply excavated. This characteristic of the muscular wasting has recently been made available for diagnosis by the excellent studies of Pottenger. In some early cases the face remains full and is thus apt to deceive as to the state of nutrition of the patient whose trunk and abdomen are markedly emaciated.

**Effects of Emaciation.**—The weakness, weariness, loss of strength and vigor of the consumptive are greatly due to the muscular atrophy even in the early stages of the disease, and one of the best signs of improvement is the regression in the muscular atrophy. There appears to be a direct relation between emaciation and the course of the disease. With each extension of the process in the lung, with each hemorrhage, he loses in weight, and with each improvement he gains in this direction, while in quiescent cases the weight remains unaltered. It may be stated that, with some exceptions to be mentioned later, the scale may be taken as a fair index of the evolution of phthisis and when we consider it in connection with the temperature curve, we can follow the case and interpret it from the prognostic standpoint with a fair degree of safety.

There are, however, exceptions: Patients in whom the disease has been arrested, *i. e.*, in whom a quiescent lesion is smouldering, are apt to remain underweight indefinitely, though they feel quite well and are more or less efficient.

When patients are progressively losing it is not advisable to tell them the extent of their denutrition. The discouragement often pulls them down much further. Conversely, it is often observed that patients gain weight after changing their physician, entering a new

sanatorium, etc., and thus gain a false impression that they are on the road to recovery. But after the novelty of the new surroundings has worn off, the gain ceases. They may then even lose progressively, and finally weigh less than before admission to the institution. To be of favorable prognostic significance, gain in weight must be persistent for several months.

In some cases of phthisis the emaciation is rapid and extreme, within a few months the body of the victim is reduced to a skeleton. These are the cases in which the disease runs an acute and progressive course—galloping consumption. Now and then we meet with patients in whom the disease is chronic, lasting for many years, still the emaciation is severe; the ribs, robbed of their adipose covering, protrude between the atrophied intercostal muscles so that we are unable to adapt the bell of the stethoscope to the chest. This cachectic form of phthisis is mostly seen in old people and, inasmuch as they have no fever and hardly cough, latent cancer is at times erroneously diagnosed.

**Prognostic Significance of Emaciation.**—Sanatoriums advertising their advantages usually show the average number of pounds gained by the patients during a certain period, and patients usually gauge their progress by the scale. This is correct in the vast majority of cases. An improving patient is one who gains in weight, and one who loses progressively is doomed. But to this there are some exceptions. Gains in institutions while the patient is under a rigorous rest cure and overfed for long periods are good as far as they go. But in order that the patient should be pronounced improved or cured it is necessary that he should hold his gains after he becomes active at his occupation or at some other vocation which suits him. In this regard, the graduated labor system of Paterson at Fromley is superior to other forms of institutional treatment. The gains attained at Fromley are more lasting than those in the institutions where the inmates lead a lazy or indolent life. Similarly, patients who are treated at home and allowed to do some work while under treatment are more likely to keep their gains than the former class.

We must be careful in evaluating gains in weight. Sometimes the patient keeps on gaining moderately while the disease is progressing and we wonder why this is so. A careful investigation may show that the lower limbs are edematous, and it is not fat and flesh which is responsible for the increase in weight, but dropsical fluid.

At times we meet with patients in whom the lesion in the lungs is improving or stationary and they have a good or even a voracious appetite, yet they keep on losing in weight. This is usually due to intestinal tuberculosis in which there may not be the characteristic diarrhea. This is a diagnostic point worth remembering because it is often very difficult to decide whether the intestine is implicated in the process, and the prognosis depends so much on the condition of the bowels.

**Seasonal Influences.**—The seasonal influences on the weight of consumptives are best studied in sanatoriums. It appears that there are significant differences in this regard. Minor,<sup>1</sup> in Asheville, reports that the chief gains are noted during the months of October to May, falling off during the summer. At North Reading, Mass., Burns<sup>2</sup> found that the minimum amount of weight loss occurs in the colder months; the maximum loss occurs in the warmer months; and rapid increase in amount of emaciation appears during the spring months. Going hand-in-hand with this is the fact that deaths in July outnumber all other months. At the Adirondack Cottage Sanitarium, Brown<sup>3</sup> found that the weight curve in pulmonary tuberculosis, if not influenced by change of climate or some other factors, rises from August to Christmas (sometimes to November), remains more or less stationary with minor fluctuations from Christmas to Easter (March), and sinks gradually from Easter to August. Brown adds that this corresponds closely to the normal weight curve. Among private patients in New York City I find that the summer months are not conducive to gains in weight, nor are the autumn months with their variable weather; but during the winter, especially during very cold seasons, the gains are extraordinary; even patients who are running low from one reason or another often gain somewhat or remain stationary during December, January, and February.

This is not true of other climatic regions. In a careful study of the weights of consumptives in eight sanatoriums in Denmark, N. S., Strandgaard<sup>4</sup> found that weekly weighing shows low gains during the winter and spring months from December to May. Then there is a distinct rise during the summer months, June, July and August, reaching its maximum in September and declining in October and more so in November and reaching its minimum in December. This is the exact opposite of conditions in the United States.

The subject deserves careful study in connection with meteorological conditions.

**Fat Consumption.**—The term "fat consumption" may appear incongruous, but we meet with cases of active phthisis in which the panniculus adiposus is well preserved, or even with excessive actual obesity, the *phthisiques gras* of French writers. I see several cases of this sort annually in my private and hospital work. They appear healthy, with rosy cheeks and well-formed bodies and their only trouble is that nobody believes that they are tuberculous. They cough and expectorate, often profusely, quantities of sputum reeking with tubercle bacilli, run a mild subfebrile temperature, at times have night sweats. Many have more or less profuse hemoptysis and in two that were under my care the cause of death was copious terminal hemorrhages.

<sup>1</sup> Kleb's Tuberculosis, p. 174.

<sup>2</sup> Boston Med. and Surg. Jour., 1914, clxx, 564.

<sup>3</sup> Osler's Modern Medicine, i, 380.

<sup>4</sup> Beitr. z. klin. d. Tuberk., 1914, xxxii, 179.

When these patients present themselves for examination one is loath to make a diagnosis of phthisis even when physical exploration of the chest reveals a typical lesion in one or both lungs or cavitation which is not uncommon. The course of the disease is rather slow; we may follow them for years without noting any marked changes in their general condition despite the fact that the lesion in the lungs is progressing and excavations are forming. Of course, only positive sputum findings are convincing in these cases.

The obesity is mostly seen in female consumptives, though I have met it in males, especially alcoholics and those having a history of syphilis. They usually have a voracious appetite and when told that they must eat well, they follow directions, often overdoing it. Combined with the rest which is urged, the overfeeding is effective in producing fat despite the activity of the disease. In tuberculosis implanted on pulmonary emphysema, and also in fibroid phthisis the weight of the patients is often above the average, though obesity is observed only rarely.

Fat consumption is also observed in children, especially infants of tuberculous stock. They appear well nourished and fat, but when we examine their muscles we find them flaccid and soft. These "pasty" infants have no resistance against infection and are carried off by any acute disease which flares up the dormant tuberculous lesions. Similarly, tuberculous meningitis and bronchopneumonia are often seen in rather fat children.

### THE SKIN.

In addition to the wasting of the muscles and subcutaneous fat, atrophy of the skin is one of the early changes in phthisis. Wheaton and, especially, Pottenger, have studied this symptom in great detail. On inspection it is noted that the skin over the site of the lesion is thin, and the subcutaneous tissue vanished. According to Pottenger, this is part and parcel of the general degeneration, and occurs after the process has existed for some time. It denotes chronicity rather than earliness, although it is often found over comparatively early tuberculous processes. In such cases it may be presumed that there was an old quiescent lesion which has become the seat of renewed activity.

The complexion of the consumptive is usually pale, though at times we meet with patients advanced in the disease who have retained a florid color. In some the hectic flush is evident at first sight; it is mostly seen at the time when the daily rise in temperature occurs. Occasionally this redness appears only on one cheek, corresponding usually to the affected side of the lung, as is discussed elsewhere. In fibroid phthisis, and in those with emphysema, there may be cyanosis of variable degree. In many cases with extensive excavations in both lungs there is hardly any cyanosis, at most some livid tint of the lips may be elicited on careful observation, but in fibroid phthisis the

cyanosis is frequently marked. In far-advanced disease with amyloid changes, the skin shows the characteristic appearance of this condition.

**Cholasma Phthisicorum.**—Smooth, shining, and non-desquamating, yellowish-brown spots are occasionally seen quite early in the disease on the forehead and upper parts of the face. They are frequently single, but often confluent, forming large patches which in female patients may be a great source of annoyance. My experience with consumptives confirms the observation made long ago by Jeanin to the effect that cholasma phthisicorum is mostly seen in connection with enlarged glands and that these patients only rarely suffer from hemoptysis. In fact, I have looked in all cases of hemorrhage that have come under my observation during the past five years and found no one with this eruption of the skin, while among my other patients it is quite frequent. In advanced cases we often meet with brownish coloration of the skin, mostly marked on the face, but at times all over the body, simulating the smoky gray or bronze color seen in Addison's disease. Considering the frequency with which the adrenals are found affected in consumptives, we have an explanation for this phenomenon.

Patients who sweat profusely may show miliaria or sudamina on the chest and abdomen. Herpes zoster of the trunk and limbs may also occur, mostly in patients with caries of the spine.

**Pityriasis Tabescentium.**—In more or less advanced cases other skin eruptions are often seen which are, within certain limits, characteristic of phthisis. In those who sweat profusely the atrophied skin is, during the day, dry, pale and brittle, and the upper epidermic layer desquamates and sheds yellow or gray scales. In some cases it looks as if the skin was covered with dust. It is known as pityriasis tabescentium and occurs mostly in consumptives who are not extremely emaciated, but who have excessive secretion of sweat and sebum; it is localized over the chest anteriorly and posteriorly, but at times the entire body is covered with it. It may be seen in other wasting diseases, but most often in phthisis.

**Pityriasis Versicolor.**—This is even more often seen in phthisis. The eruption is discretely scattered over the anterior and posterior aspects of the thorax and consists of small macules, slightly raised above the level of the skin, round or oval in shape with well-defined margins. Scales can be scratched off and when examined show roundish, shining microscopic spores, the *Microsporon furfur*.

The color of the eruption varies in different individuals, but is mostly brown or a dirty yellow, darker in those who lead an outdoor life, and over the arms and neck when these are affected, while in negroes they are almost white. In patients who neglect to attend to cleanliness of their bodies the macules may coalesce, forming large, irregular plaques covering large tracts of skin anteriorly and posteriorly, which desquamate upon scratching.

It is seen in consumptives who sweat profusely at night, which

favors the growth of the fungi, and in patients whose skin has a tendency to scale which assists in their detachment. Piéry<sup>1</sup> has inoculated guinea-pigs with the scales removed from such patients and obtained positive results, and he suggests that it is a tuberculous dermatomycosis.

When seen on the chest, pityriasis versicolor is fairly indicative of phthisis, although it occurs in other cachectic diseases, notably cancer.

We also meet with acnitis and folliclis, characterized by the eruption of red or dark brown nodules over the face, and more often over the back between the shoulder-blades and over them. We find these nodules in various stages of development, some becoming pustular and when the pus is discharged an ulcer remains, which heals, leaving a scar. They are found in exceedingly chronic cases. It has been my impression that the administration of creosote and arsenic and their derivatives is effective in enhancing these eruptions.

**The Hair.**—Many authors have stated that alopecia is more frequent in phthisical subjects than in others, and it has been attributed to the same causes as those acting when the hair falls out after an attack of typhoid fever, etc. But in my experience this is not true. The tuberculous patients in my hospital and private practice are not more often bald than others of the same class, nor do I meet with many consumptives who have localized alopecia, or alopecia areata. Premature grayness of the hair, which Cornet mentions as very frequent among consumptives, has also not been found by me to be frequent in tuberculous patients in the United States.

**Clubbed Fingers.**—Clubbed fingers were already mentioned by Hippocrates as a symptom of phthisis, and French writers at present call them *doigts hippocratiques*. They are found in about one-third of advanced consumptives, and are probably caused by chronic peripheral passive congestion. Clubbed fingers are not exclusively met with in phthisis, but also in empyema, bronchiectasis, chronic bronchitis, asthma and pulmonary emphysema, in thoracic aneurisms, etc. They have also been encountered in rare cases of cirrhosis of the liver and amyloid disease.

In phthisis we usually find that the fingers of both hands are thickened and bulbous, like a club or drumstick, resembling somewhat the condition seen in chronic onychia. The terminal phalanges are enlarged, the nails curved longitudinally and laterally. From radioscopic studies it appears that the bones and joints are not affected, nor is the skin altered in any way, but only the superficial soft parts are hypertrophied. As to what the change consists in we are in ignorance because of lack of anatomical and histological studies. Some have suggested that it is a fibrous thickening of the innermost layers of the epidermis, as a result of prolonged congestion of the capillaries. This may be true of some cases, but in those in which

<sup>1</sup> Gaz. d. hôpit, 1912, lxxxv, 531.

the condition develops within a few weeks it is doubtful whether this could be the actual anatomical change.

In most cases the onset is slow and insidious and the patient knows nothing about it till the physician calls his attention to the clubbed

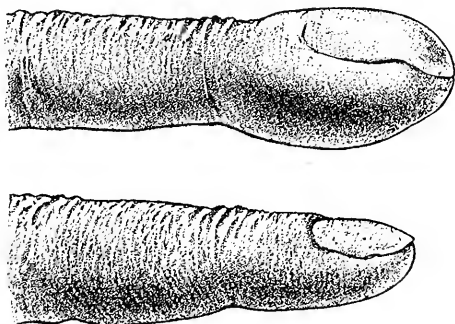


FIG. 32.—Clubbed fingers and curved nails.

fingers. But on rare occasions, as has already been noted by Trousseau, it comes on very quickly and within a few weeks the fingers look like drumsticks. In these acute cases they may be painful, tender and livid. Lividity is also seen in those suffering from pulmonary emphysema or fibroid phthisis. The nails are curved and look like claws.



FIG. 33.—Clubbed fingers in phthisis.

My observations are in agreement with those of Bezançon<sup>1</sup> that clubbed fingers are not met with in all cases of chronic phthisis, as

<sup>1</sup> Arch. gén. de Médecine, 1904, i, 1663; ii, 3100.



FIG. 34.—Changes in the toes in tuberculous osteo-arthropathy.

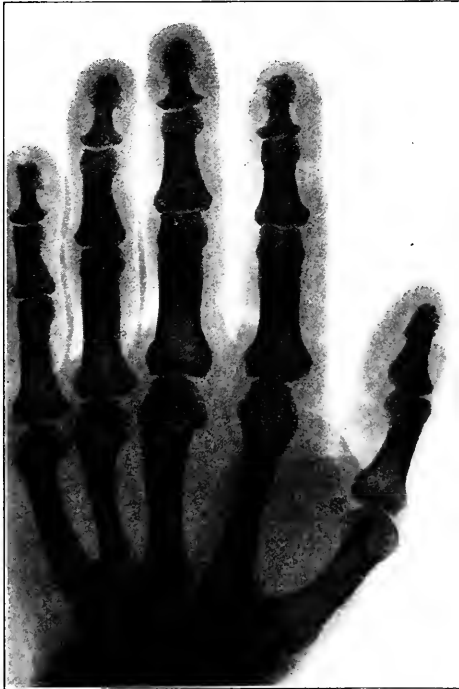


FIG. 35.—Radiogram of a hand in a case of clubbed fingers in pulmonary osteo-arthropathy with bronchiectasis and pulmonary emphysema. On the tips of the end phalanges marked cauliflower formations; bony excrescences on basal portions of some phalanges; typical Heberden's nodes; broadening of the bases of the middle phalanges.



some have stated. A large number of consumptives have normal-shaped fingers, while some have even long, tapering terminal phalanges. Clubbed fingers are encountered almost exclusively in fibroid phthisis, pulmonary emphysema with tuberculosis and in those having extensive pleural adhesions. In other words, whenever clubbed fingers are encountered in a case of phthisis we find that the patient is also suffering from dyspnea and dilatation of the right heart. This would suggest mechanical disturbances of the circulation, causing peripheral



FIG. 36.—Radiograms of hand in a case of fibroid phthisis.

venous stasis. Moreover, the prognosis in these cases is quite favorable as regards duration of life, though the outlook as to comfort is rather gloomy.

**Pulmonary Osteo-arthropathy.**—In some chronic cases we meet with enlarged hands simulating those seen in acromegaly. The fingers are altogether increased in volume, the nails enlarged and curved like the beak of a parrot. The metacarpophalangeal region is usually normal, but the wrist is enlarged and deformed, bulging on its dorsal aspect. In many cases there is also some deformity of the spine—kyphoscoliosis, and the feet may show the same changes as the wrists and hands, especially the toes and tarsus. In the cases

that came under my observation there were pains of variable severity, sometimes unbearable and generally intermittent. As can be seen from the radiograms (Figs. 35 and 36) the differences between pulmonary osteo-arthropathy and simple clubbed fingers consists in this: In the former the bones and joints are hypertrophied and some osteophytes may be seen at the line of the joint cartilages, while in the latter only the soft parts are implicated, the bones remaining practically normal. In his recent thorough study of this subject, Edwin A. Locke<sup>1</sup> is inclined to regard clubbed fingers in phthisical patients as identical with osteo-arthropathy, the former representing an early stage of the latter. He also found with clubbed fingers early proliferative changes in the periosteum of some of the long bones of the forearm and lower legs of exactly the same type as in hypertrophic osteo-arthropathy. Clinically we distinguish these two conditions by the fact that in clubbed fingers only the terminal phalanges are enlarged, while in osteo-arthropathy the wrist is also affected, and the feet usually show the same changes and in addition there is in most cases decided spinal deformity. But this does not exclude the identity of the two processes if we choose to regard clubbed fingers as the early stage of osteo-arthropathy. The former is, however, far more common.

<sup>1</sup> Archives of Internal Medicine, 1915, xv, 659.

## CHAPTER XII.

### SYMPTOMS REFERABLE TO THE CARDIOVASCULAR AND RENAL SYSTEMS.

#### THE CARDIOVASCULAR SYSTEM.

**Cardiac Palpitation.**—Of the functional cardiovascular disturbances in phthisis the most important are palpitation, tachycardia, and hypotension. They are very often associated, but at times we meet one to the exclusion of the other.

In incipient cases palpitation is mainly met with in young persons, especially chlorotic girls. Slight or moderate exertion, excitement and emotional disturbances may cause an attack, or it may occur without any provocation. At times it is very pronounced, and is perhaps the only subjective symptom which induced the patient to consult a physician. Rarely it is very severe and is accompanied by precordial pains and distress and by vasomotor disturbances such as pallor or flushing of the face, sweating, etc.

I have met with cases in which palpitation preceded all subjective and objective symptoms of incipient phthisis. Some are for this reason treated for heart disease. Recently I saw a case which was treated for hyperthyroidism, but careful examination showed an incipient lesion of the left apex.

The causes of the palpitation at this stage are not clear. Some have been inclined to attribute it to dilatation of the right heart, but we meet it in cases in which this organ is normal. Others believe it is due to the anemia—low arterial tension—or to sympathetic nerve disturbances. The last factor is apparently operative in many cases, because we meet it mostly in nervous patients, in young girls and in women during the menopause. Compression of the vagus by enlarged glands may be the cause in some cases.

Cardiac irritability is seen also in advanced but quiescent cases. The patient is doing well, has no fever, no cough and is not emaciated. But the least exertion, emotion, or complication provokes cardiac distress which may be very painful, almost anginal. Here, the palpitation is due to cardiac dislocation and occurs more often in left-sided lesions. A large cavity in the left lung with pulmonary contraction has drawn the mediastinum to the left and the diaphragm upward, so that the heart is pushed upward and to the left and the apex beat may be found in the third interspace at the axillary line. In a recent case of this character I also found arrhythmia. The palpitation is not

so pronounced in right-sided dislocations of the heart, not even in complete dextrocardia.

Palpitation has no influence on the course of phthisis excepting in the advanced stages when it is due to dislocation of the heart. In the early cases we may meet with annoying palpitation in nervous patients who are progressively improving. But from the diagnostic standpoint it is a symptom of great value. Hirtz said that "when a patient complains of palpitation, examine his lungs; and examine his heart when he complains of dyspnea." While this does not hold good in every case, yet it is well worth bearing in mind, especially when dealing with an anemic youth. In some cases of phthisis we meet with palpitation for a day or two before the occurrence of hemoptysis.

**Tachycardia.**—Rapid heart action objectively ascertained—which may not be known to the patient at all, thus differing from palpitation, which is a subjective symptom—is very frequent in all stages of phthisis. In my experience, over 80 per cent. of cases of incipient phthisis have tachycardia which is usually permanent or, rarely, paroxysmal. It is a symptom of phthisis which is not appreciated to the extent it deserves, though it is often very helpful in deciding a doubtful case.

The tachycardia may be of toxic origin. Every elevation of temperature in phthisis, as in other conditions, is accompanied by an acceleration in the pulse rate. But it is often pronounced in those running a subfebrile temperature and also in afebrile cases. In fact, in tuberculosis the pulse is accelerated far out of proportion to the height of the temperature. In most other cases an elevation of 1° F. is usually accompanied by an increase in the pulse rate of about eight beats per minute, while in phthisis we often have a temperature of 100° while the pulse counts 120 and even more. In fact, in most afebrile cases of phthisis the pulse is over 90 per minute and during the morning subnormal temperature tachycardia is not at all rare. Thus tachycardia is an early symptom of phthisis and some writers consider it a premonitory symptom.

**Permanent Tachycardia.**—In a large proportion of cases the tachycardia is permanent and accompanied by subjective discomfort, such as palpitation, languor, debility, dyspnea, etc. In others, it is purely objective, the patient is hardly aware of its presence. I have observed many cases in which the disease was arrested or even cured, yet the tachycardia remained. At times it greatly interferes with the patient's efficiency. But I cannot agree with Minor who says that in an arrested case one cannot feel safe as to the continued progress of the patient so long as the pulse rate remains high. I have seen patients who have been able to work for a living without much discomfort in spite of the rapid heart action.

One characteristic of the pulse of the consumptive is its instability and variability. While resting the rate may be normal, but the

slightest exertion—a fit of coughing, some emotional experience, a heavy meal, or changing from the reclining to the erect posture—may send up the pulse rate to 110 or 120, though Wells says the contrary and Minor found it present as often as absent; Faisans says that he does not know of any disease in which the pulse is as unstable as in phthisis.

**Paroxysmal Tachycardia.**—In rare cases we meet with paroxysmal tachycardia. The patient feels comparatively well and, without any exciting cause, he is seized with severe palpitation, dyspnea, or even orthopnea, and cyanosis. Counting the pulse rate, we find it 150 to 200 per minute, small, wiry and often irregular. The attack may last a few hours, a day or two. In one case the patient got an attack while in my office, the pulse going up from 96 to 160, and looked as if he was breathing his last. He recovered in two hours.

After several attacks, which may come on at frequent intervals, we may observe signs of cardiac dilatation—the heart gives way and the result is edema of the lower extremities, enlargement of the liver, etc. Finally, asystole occurs and the patient succumbs. Paroxysmal tachycardia is of grave significance and, when occurring several times, will ultimately kill the patient during one of the attacks.

**Causes of Tachycardia.**—The causes are obscure. It has been attributed to bulbar lesions, to interstitial neuritis of the pneumogastric nerve and to myocarditis, etc. Some believe that it is due to compression of the vagus by enlarged tracheobronchial glands, but it would seem that the effect should rather be a slowing of the pulse rate than an acceleration. Indeed, considering that the vagus is often pressed upon by enlarged glands, it is noteworthy that a slow pulse is exceedingly rare in phthisis. Other authors have attempted to explain this phenomenon by stating that it all depends on which part or branch of the pneumogastric is affected by the tuberculous process. On this also depends whether the stomach or myocardium will suffer. K. Bohland<sup>1</sup> is inclined to ascribe the tachycardia in phthisis to the small heart characteristic of the disease—in order to pump enough blood into the system, the heart must beat more often. In the advanced stages of phthisis it is due to myocarditis. The tuberculous toxemia alone does not explain the tachycardia because it is found often in afebrile patients, as was already stated.

Permanent tachycardia aggravates the prognosis of phthisis and these patients should not be sent to a high altitude. The causes are complex and vary with each case. In patients in whom it is of toxic origin we may expect improvement as soon as the fever subsides. But in many it is caused by compression of the pneumogastric nerve by enlarged tracheobronchial glands, neuritis of that nerve, or reflexly of gastric origin, fibrous degeneration of the cardiac muscle, or tuberculosis or hyperfunction of the adrenals, etc. When due to cardiac displacement, especially to the left in left-sided lesions, it is permanent.

<sup>1</sup> Brauer, Schröder, and Blumenfeld's *Handbuch der Tuberkulose*, 1915, iv, 4.

Arrhythmia is only rarely observed in phthisis and the prognosis of these cases is rather unfavorable.

**Bradycardia.**—A slow pulse is exceedingly rare in phthisis; those who see large numbers of these patients occasionally meet one with a pulse less than 50 per minute. One case under my care had a pulse rate of 36 per minute for several months, and only during febrile attacks did it rise to 50 or slightly more. Gueneau de Mussy, who described some of these cases, attributed it to irritation of the pneumogastric nerve. On the other hand, there are many physicians of large experience who have never seen bradycardia in phthisis. From the few cases met by me, it appears that the prognosis in cases with a slow pulse is very good. "A slow pulse when met with is always of good augury," says Sir Douglas Powell,<sup>1</sup> "and in some obscure and borderline cases it would tell with considerable force in favor of the case not being one of tuberculosis."

At the terminal stage of far-advanced phthisis we often meet with a slow, soft, almost imperceptible pulse which intermits, indicating cardiac failure or exhaustion. The pulse is also slowed when meningeal irritation complicates the disease.

**Arterial Hypotension.**—The blood-pressure, measured with a sphygmomanometer, is lower than normal in the vast majority of phthisical patients. It is evidently due to the toxic effects of the metabolic processes of the tubercle bacilli, because an injection of tuberculin is usually followed by a decided fall in the blood-pressure. Sir Douglas Powell says that the large doses of tuberculin which were used in the first days of Koch treatment of lupoid and other forms of tuberculosis caused severe collapse, and recent writers, like Levy, Geisbock and others found that, even in small or moderate doses, tuberculin reduces arterial tension. It has been found that a low blood-pressure is an almost constant characteristic of the very early stages of phthisis and, when occurring in an adult without any other assignable cause, tuberculosis is to be suspected. John Ritter<sup>2</sup> found hypotension in cases of phthisis before the physical signs and even before elevation of temperature were definitely demonstrable. My own experience has brought me to the conclusion that in cases presenting obscure symptoms and signs of phthisis, when accompanied by a low blood-pressure, the diagnosis may be safely made; conversely, I always hesitate in cases with high arterial tension, excepting in persons over fifty years of age. But even in these high pressure is exceedingly rare in phthisis.

Sir Douglas Powell showed that slight exercise raises the blood-pressure in quiescent cases, while in acute cases it is lowered (Figs. 37 and 38).

This hypotension is quite marked in the early stages and becomes more accentuated with the progress of the disease. I find that, as

<sup>1</sup> *Lancet*, November 23, 1912, p. 1415.

<sup>2</sup> *Trans. Nat. Assn. Study and Prev. Tuberc.*, 1911, vii, 297.

a rule, cases of undoubted phthisis with a normal or high blood-pressure have a favorable prognosis. When the blood-pressure is low at first but rises gradually, it is an excellent indication of improvement; conversely, tuberculous patients with normal or high blood-pressure who begin to show hypotension almost invariably also show indica-

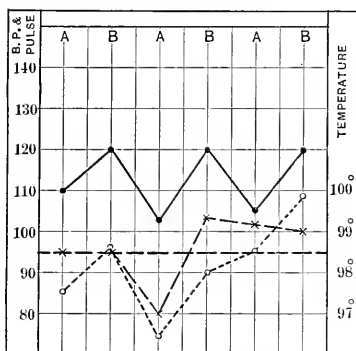


FIG. 37.—Chart of blood-pressure in a case of quiescent tuberculosis before and after exercise. Blood-pressure, ●— (A, before exercise; B, after exercise). Pulse rate, ○ . . . . Temperature, x----- (R. Douglas Powell.)

tions of the extension of the process in the lung and the prognosis is aggravated. I have not noted in many cases any relation between the hypotension of phthisis and the temperature, the pulse rate, or the dyspnea. It is met with in febrile and afebrile cases; in young and in the aged.

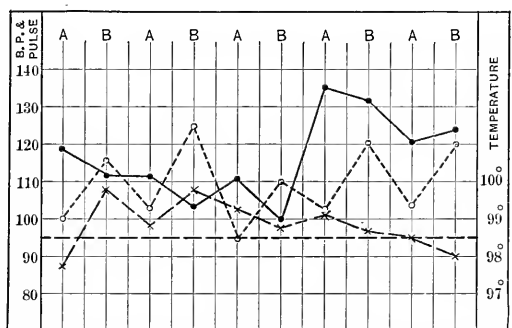


FIG. 38.—Chart of blood-pressure in tuberculosis before and after exercise. Blood-pressure, ●— (A, before exercise; B, after exercise). Pulse rate, ○ . . . . Temperature, x----- (R. Douglas Powell.)

It has also been observed by many authors that patients with a tendency to hemoptysis have a high blood-pressure which rises before the onset of the bleeding. At one time I tested this point in several patients but could not confirm it. Many who bled profusely had a very low blood-pressure.

## THE BLOOD.

**The Erythrocytes.**—Despite the external appearance of anemia frequently seen in many phthisical patients in all stages of the disease—which has given rise to the expression “great white plague”—no changes in the cytology of the blood characteristic of the disease have been found. In fact, it is noteworthy that many patients who look pale show an almost normal blood picture. At times a polycythemia is encountered, but the hemoglobin is not increased under the circumstances. Only on rare occasions have I found a decided decrease in the number of erythrocytes, especially during the very early and very advanced stages of the disease. In some few cases the count was as low as 1,000,000, or even less, but the fact that it is so rare shows that it is an accidental occurrence and cannot be considered characteristic of the disease. After profuse pulmonary hemorrhages the anemia may be profound, but it is remarkable that the blood improves very rapidly after the cessation of bleeding.

There is very often noted a decidedly low percentage of hemoglobin in incipient cases, even when the erythrocytes are not decreased in number. For this reason some authors have spoken of a pseudo-chlorotic blood picture. But soon after the patient is placed under proper dietetic and hygienic treatment the hemoglobin content of the blood improves, as a rule. It may be stated that in many cases there is slight diminution in the number of erythrocytes, and a pronounced diminution in the hemoglobin content during the incipient and far-advanced stages of phthisis.

From the researches of Limbeck, Grawitz, and others it appears that with the advance of the disease, even with the formation of pulmonary excavations, the blood picture is very often not deviating from the normal. The yellowish pallor, “ochrodermia,” which is so frequent at this stage, is not due to alterations in the cytology of the blood, so far as can be ascertained. But there is good reason to believe that the total amount of blood in the body is less than in healthy individuals; that there is a distinct oligemia. This has been ascribed to the loss of water through profuse night sweats, expectoration, and often diarrhea, which brings about a higher specific gravity of the blood with a concentration of the cells.

In the far-advanced stages, with hectic fever, often complicated by mixed infection, there is in addition to leukocytosis, also a diminution in the number of erythrocytes, with a fall in the percentage of hemoglobin.

**Leukocytes.**—In incipient phthisis the leukocytes are quite normal in number and variety. Even in acute cases, so long as there is no mixed infection, the leukocyte count is unaffected. Some authors, notably Ullom and Craig<sup>1</sup> in this country, have found a slight leuko-

<sup>1</sup> Amer. Jour. Med. Sci., 1905, cxxx, 386.



cytosis which increases somewhat with the advance of the disease. But inasmuch as it only reaches about 11,000 to 14,000 on the average, it cannot be considered of any value diagnostically. Kjer-Petersen<sup>1</sup> found that in women the number of white-blood cells oscillates between 4000 and 25,000 under normal physiological conditions.

Gerald B. Webb, G. B. Gilbert, and L. C. Haven<sup>2</sup> found the blood platelets are increased in number in cases of phthisis. In tuberculosis in guinea-pigs they observed the same phenomenon. They believe that the blood platelets either contain or supply opsonins. The fact that they are increased at an altitude of 6000 feet would, according to Webb, point to a reason for the salutary effects of high climates on phthisical patients.

With the advance of the disease leukocytosis is not rare; it is usually transient, but rarely permanent. It appears to depend on the activity of the tuberculous process, the intensity of the fever, the presence of complications, etc. But there are so many exceptions to this rule that it cannot be utilized for diagnostic and prognostic purposes. It appears, however, that an injection of tuberculin is usually followed by transient leukocytosis. Some have attempted to judge the presence of excavation by the white-cell picture, but have failed. Wright's attempt to utilize his tuberculo-opsonic index in the prognosis of tuberculosis has also failed to give satisfaction to most authors.

**Arneth's Blood Picture.**—A great deal has been made during recent years of Arneth's blood work in infectious diseases, especially tuberculosis. His theory is based on his observations of the growth of the neutrophile and the changes of the nuclei, or granules within these cells during the period. He developed a very complicated blood picture based on the number of granules or fragments in each neutrophile. His contention is that when the disease takes a bad turn there is an increase in the number of young forms of neutrophiles containing but one granule as a nucleus, and a decrease in the older forms of cells which correspond to the polymorphonuclears of other writers; he calls it a shifting of the blood picture to the left.

Arneth's work has been tested by many other authors and but very few have been able to confirm his contentions that the changes in the blood picture go hand-in-hand with the clinical course of the disease, nor have many agreed with his interpretation of the origin of the changes in the neutrophiles. In this country some authors have found Arneth's blood picture of value in diagnosis and prognosis, especially Minor and Ringer,<sup>3</sup> and James Alexander Miller and Margaret A. Reed.<sup>4</sup> Miller, in an exhaustive study of the leukocytes in tuberculosis, arrives at the conclusion that it gives valuable information as to the prognosis and clinical course of phthisis, but in the diagnosis of

<sup>1</sup> Brauer's Beiträge, 1906, Beiheft.

<sup>2</sup> Arch. Intern. Medicine, 1914, xiv, 743.

<sup>3</sup> Amer. Jour. Med. Sci., 1911, cxli, 638.

<sup>4</sup> Arch. Intern. Med., 1912, ix, 609.

incipient cases it is of no value. In his experience a leukocytosis, an increased percentage of small lymphocytes, a diminished percentage of eosinophiles and a marked shifting of Arneht's blood picture to the left are characteristic of cases of pulmonary tuberculosis which are progressively doing badly, or an exacerbation of the disease.

I have given this method a trial and could find no diagnostic or prognostic hints which were constant; in fact, the contradictions were so frequent and notorious that I have abandoned it altogether. Pappenheim, Politzer, Hiller, and, in this country, Solis Cohen,<sup>1</sup> and Strickler and Kagan<sup>2</sup> have arrived at the same conclusion.

**Tubercle Bacilli in the Circulating Blood.**—During recent years many investigators have found tubercle bacilli in the circulating blood of patients suffering from phthisis. Some have found them in the blood of patients with advanced forms of the disease, while others have even detected them in early cases. Rosenberger,<sup>3</sup> Koslow, Kurashige, and others have even stated that in all cases of tuberculosis, bacilli may be found when carefully looked for, while P. Klemperer found them in 7 cases in which the disease was only suspected, but could not be diagnosed with the usual clinical methods. But when still others, like Liebermeister, Suzuki and Takaki, and Kurashige discovered tubercle bacilli in the blood of apparently healthy individuals, and Clara Kennerknecht in the blood of 91 per cent. of 120 healthy children of which only 68 were tuberculous, the hopes entertained that we might have in this a good method of discovering tuberculosis as a bacteremia before the onset of clinical symptoms began to vanish. The history of tuberculin as a diagnostic agent was here repeated.

Further investigations by Walter V. Brehm,<sup>4</sup> Beitzke, Schern and Dold have shown that there was a source of error: The tap water used in diluting the blood often contains acid-alcohol-fast rods which look like tubercle bacilli under the microscope. These acid-fast rods may be bacilli or some other substances, but they are not pathogenic to guinea-pigs. It has also been found that fragments of red-blood corpuscles may take on the stain of the tubercle bacillus and show acid-fast properties.

These findings were verified in another way. The blood of tuberculous patients was injected into animals with a view of ascertaining the proportion that would be infected with tuberculosis. The results of some authors like Anderson,<sup>5</sup> Rumpf,<sup>6</sup> Ravenel and Smith,<sup>7</sup> Querner,<sup>8</sup>

<sup>1</sup> New York Med. Jour., 1910, xcii, 248.

<sup>2</sup> Boston Med. and Surg. Jour., 1910, cxvii, 709.

<sup>3</sup> Amer. Jour. Med. Sci., 1909, cxxxvii, 267.

<sup>4</sup> Jour. Amer. Med. Assn., 1909, liii, 909.

<sup>5</sup> The Presence of Tubercle Bacilli in the Blood in Clinical and Experimental Tuberculosis, Hygienic Labor. Bull., No. 57, 1909.

<sup>6</sup> Münch. med. Wehnschr., 1912, lix, 1951.

<sup>7</sup> Jour. Amer. Med. Assn., 1909, liii, 1915.

<sup>8</sup> Münch. med. Wehnschr., 1913, lx, 401.

Leo Kessel,<sup>1</sup> and others were entirely negative—none of the animals experimented on showed any tuberculous lesions, while others got a few positive results. Liebermeister, on the other hand, found that in 6 cases the animals were infected with tubercle bacilli in the blood from human beings who showed no clinical symptoms of the disease. Others, like Kessel, found that a few of the animals inoculated with blood from tuberculous patients became tuberculous.

It was necessary to explain the presence in the blood of many cases of phthisis of bacilli, which are but rarely pathogenic to animals. It was suggested that while inoculating the animals with the blood, antibodies are also inoculated, or that the germs circulating in the blood lose their virulence owing to the bactericidal action of the blood.

At the present state of our knowledge the following conclusions of Klemperer<sup>2</sup> are justified:

Acid-fast rods are found microscopically in small numbers in the blood of a large proportion of consumptives. Animal experimentation shows that but few patients have virulent tubercle bacilli in their blood. But it must be mentioned in this connection that in order to infect a guinea-pig a certain number of tubercle bacilli is necessary, having a certain virulence, perhaps greater virulence than the bacilli that survive the bactericidal action of the blood of the average patient possesses. Negative outcome of the inoculation, for this reason, does not mean absence of the bacilli from the blood. Inasmuch as the acid-fast rods are found microscopically only in the blood of tuberculous and not of healthy persons, the negative outcome does not speak against their being tubercle bacilli. Finally, inasmuch as the frequency of the occurrence of the bacilli in the blood is supported by clinical and anatomical facts, we are justified in considering these acid-fast rods as tubercle bacilli.

The finding of these bacilli in the blood is of no potential diagnostic and prognostic value, while about their immunizing effects we cannot speak with any degree of certainty.

### THE RENAL SYSTEM.

**The Kidneys.**—There appear to be no changes in the structure and functions of the kidneys which can be considered specific and characteristic of early phthisis, excepting in cases with a very acute onset with high fever which affects these organs in the same manner as hyperthermia due to other causes, or in cases in which the kidneys are inoculated at the onset together with many other organs, as in acute miliary tuberculosis.

Some writers, notably the French, have described polyuria, phosphaturia and albuminuria as very frequent in early and even in latent

<sup>1</sup> Amer. Jour. Med. Sci., 1915, cl, 377.

<sup>2</sup> Ztschr. f. klin. Med., 1914, lxxx, 88.

phthisis. Barbier<sup>1</sup> says that albuminuria is often the only sign observed for a long time before other symptoms make their appearance; and that this albuminuria is often misunderstood by physicians. Albert Robin<sup>2</sup> describes pretuberculous polyuria: The quantity of urine in the early stage is increased; in the second stage normal; and in the third stage diminished, although some patients have polyuria throughout the course of the disease. The oliguria of the advanced stage is closely related to the fever, sweats, and eventual diarrhea. Robin maintains that the polyuria of early phthisis is simple, showing no abnormal constituents or, at most, there may be phosphaturia, when, at times, it may be severe enough to cause irritation of the kidney substance, congestion, and, finally, albuminuria.

These changes have, however, not been met with sufficient constancy to place them in the category of pathognomonic or specific symptoms of early tuberculosis. Among 100 cases of early tuberculosis that I have especially investigated for the purpose of testing this point, I found albuminuria in only 9 cases, and casts in only 3.

**Albuminuria in Advanced Cases.**—In the advanced stages albuminuria is very frequent. Montgomery found albumin present in about one-third of cases of phthisis. In the majority of cases the amount was only a trace and when found in larger amounts it was always associated with casts and blood or pus. It appears that cases with intestinal ulcers have larger amounts of albumin than others. From his studies he arrives at the conclusion that a large number of casts in the urine of consumptives is indicative of an unfavorable prognosis, and the reverse.

As to the causes of the albuminuria we are not clear. Some look upon it as caused by the irritation of the tuberculous toxins, which are eliminated with the urine, on the renal parenchyma, while others see in it the effects of the chronic fever, or actual tuberculosis of the kidneys. In an exhaustive study of the problem, N. Leon-Kindberg<sup>3</sup> arrives at the conclusion that the so-called "tuberculotoxins" cause no lesions in the kidneys. The presence of isolated tubercles in the kidneys explains perhaps some cases of bacteriuria.

It must be mentioned that mixed infection, such as is seen in pulmonary cavities containing in addition to tubercle bacilli also pyogenic microorganisms, is usually the cause of albuminuria in the advanced stages of phthisis where there is no concomitant renal tuberculosis.

**Nephritis in the Course of Phthisis.**—Symptoms of acute nephritis are very rarely met with during the course of phthisis; but the chronic degenerative forms, parenchymatous and interstitial have, however, been found in variable proportions. Bamberger found nephritis together with phthisis to the extent of 15 per cent.; Potain states that one-fifth of all consumptives have nephritis; and others have found

<sup>1</sup> Brouardel and Gilbert's *Traité de Médecine*, Paris, 1910, xxix, 423.

<sup>2</sup> *Traitement de la tuberculose*, p. 498.

<sup>3</sup> *Études sur le rein des tuberculeux*, Paris, 1913.

even higher percentages. Senator was inclined to the opinion that tuberculosis is an important etiological factor in chronic parenchymatous nephritis. But it appears that clinical symptoms of nephritis are usually altogether absent, even when albumin and casts are found in the urine, and cardiac hypertrophy is exceedingly rare.

Most of these views are based on the presence of albumin in the urine, and Montgomery<sup>1</sup> has shown that in pulmonary tuberculosis albumin and casts are not often associated with evidences of nephritis. In phthisis, albuminuria is not necessarily a manifestation of nephritis, or even of renal tuberculous lesions, but in many cases, especially in fibroid phthisis and emphysema, it is due to cardiac dilatation, to intestinal and hepatic disturbances, etc., which are so frequent in advanced phthisis. Albuminuria may also be the sole indication of a tuberculous lesion in a kidney which manifests itself by no other symptom during life. Thus, in a painstaking study of 106 pairs of kidneys taken from consumptives, made by J. Walsh,<sup>2</sup> 53.9 per cent. were found to contain tubercles. He also found that among these 106 pairs of kidneys only 10 showed chronic interstitial nephritis, while in 44 kidneys from patients suffering from other chronic diseases, there were 23 with this form of nephritis, which clearly indicates that tuberculosis of the lungs is antagonistic to the ordinary chronic general interstitial nephritis, just as it appears antagonistic to general sclerosis of other organs.

**The Amyloid Kidney.**—In the far-advanced stages of phthisis with large suppurating cavities in the lungs, we often encounter amyloid degeneration of the kidneys, as in cachexia due to other causes. It is usually found associated with amyloid changes in other organs, notably the liver, spleen, and intestines. But even this is not as frequent as would be expected. White found 9.2 per cent.; Walsh 6.6 per cent., and he never found it exclusively in the kidneys; Blum in only 6 per cent., but he points out that 79.2 per cent. of all amyloids were caused by tuberculosis, of which 54.4 per cent. is pulmonary phthisis.

Its symptomatology is that of amyloid disease of the liver and intestines, and because it is always associated with other changes in the kidneys, such as chronic parenchymatous nephritis, the resulting symptoms are always complex. Albumin is usually present in the urine. I find it safe to conclude, when the liver is enlarged and there is profuse diarrhea, that there is no doubt that the kidneys are amyloid. But when there is no diarrhea, there is polyuria of low specific gravity, casts and but little albumin.

**Terminal Edema.**—Edema is present in a large proportion of cases of advanced phthisis; the ankles and knees especially are thus affected during the terminal stages, but it does not always depend on the condition of the kidneys. Montgomery found no relation between edema

<sup>1</sup> Fourth Annual Report Henry Phipps Institute, 1908, p. 120.

<sup>2</sup> Trans. Sixth Intern. Congr. Tuberc., 1908, i, 347.

and the occurrence of albumin and casts in the urine, and suggests that the edema found in tuberculosis does not depend primarily on nephritis. General anasarca is often seen in far-advanced cases toward the end, and this may be a manifestation of the state of the kidneys, but when we bear in mind that in these cases we also have cardiac dilatation, it is clear that the pathogenesis is often complex.

The edema may be considered an ill omen, and I have not seen a consumptive with edematous ankles and knees survive, or even improve. It may be unilateral, sometimes one-half of the body is swollen and pitting, corresponding to the side on which the patient lies. At times we see it only in one upper extremity, due to pressure on the veins coming from the arm by tuberculous glands, or when they are implicated in the adhesive pleurisy of that side and more commonly by thrombosis of the innominate, subclavian or other veins. Phlebitis or thrombosis of the femoral, popliteal and crural veins is even more frequent (see Chapter XXVI).

**Uremia.**—Symptoms of uremia are not often met with in phthisis, but not so rarely as some authors would lead us to believe. In the advanced stages we meet at times with typical uremia which is often mistaken for meningeal infection. I have seen three cases of convulsions due to this cause. In severe dyspnea without fever, arising suddenly, uremia is to be thought of in cases with albumin and casts in the urine. Some diarrhea observed in these cases is distinctly of uremic origin, and at times we meet with pulmonary edema. They are usually very difficult of recognition and differentiation.

## CHAPTER XIII.

### NERVOUS SYMPTOMS OF PHTHISIS.

As an exquisitely chronic disease, phthisis is accompanied by many morbid manifestations of the nervous system; in fact, nearly every symptom of the disease is often influenced by the effects of the tuberculous toxins on the nervous system. The neurotic phenomena may make their appearance immediately at the outset, in some they precede the actual onset of phthisis, while most of confirmed consumptives have a psychology peculiarly their own and show symptoms of nervous aberration which cannot escape the vigilance of the observant physician.

**Neurasthenia and Psychasthenia.**—The onset of phthisis is often accompanied by symptoms simulating that syndrome which is known under the vague term of neurasthenia; indeed, many patients have been treated for neurasthenia for months before the true nature of their affection was recognized. These symptoms have been described by many authors and deserve careful consideration.

A large proportion of incipient and confirmed consumptives complain of vertigo, headache, pains along the spine, irritability of temper, insomnia, not necessarily due to night sweats, and fleeting pains of the chest which at times cannot be attributed to circumscribed pleurisy; frequent attacks of tachycardia, irrespective of the temperature and cardiac palpitation are not rare. There is also the characteristic languor and persistent weariness, which is not relieved by sleep; on the contrary, many state that they feel more weary and tired in the morning on getting out of bed, and that this tired feeling wears off in the afternoon or evening, all of which is suggestive of neurasthenia and psychasthenia. Considering these symptoms there is little wonder that many patients are treated for "nervousness" until an attack of dry or moist pleurisy, or of hemoptysis or a careful examination of the chest, reveals the true state of affairs. Papillon<sup>1</sup> goes so far as to say that he suspects every victim of neurasthenia to be a subject of latent tuberculosis, and G. D. Head<sup>2</sup> considers a considerable proportion of neurasthenics as harboring a tuberculous infection which is so concealed that it escapes detection by the usual clinical methods. Considering that neurasthenia is quite often the result of toxic causes, it is clear that tuberculous toxemia may be a cause of these symptoms in many cases. If the chests of all patients treated for neurasthenia

<sup>1</sup> Arch. de Scien. Médicales, 1900, v, 19.

<sup>2</sup> Jour. Amer. Med. Assn., 1914, lxiii, 996.

were carefully examined, a large proportion of phthisis which is now only recognized in the advanced stages would be identified at earlier stages.

**Reflex Nervous Phenomena.**—Aberrations of the sympathetic nervous system are not rare in phthisis. Among these may be mentioned the unilateral flushes of the face and occasionally of one ear, combined with a feeling of warmth, sweating, etc. In some cases it has been observed that the cutaneous temperature is higher on one side of the chest. These unilateral symptoms are usually found on the side corresponding to the affected hemothorax and, in bilateral lesions, to the side in which the recent or more active lesion is located. In some patients with extensive excavations in the lung, the nostril corresponding to the affected side is widely dilated. Dermographism is very frequent.

An important symptom of phthisis is dilatation of the pupils, to which Rogue,<sup>1</sup> Destrée,<sup>2</sup> and also T. F. Harrington<sup>3</sup> drew attention. Harrington described the widely dilated pupils as "not a paralyzed pupil, but rather one which seems to be in a more or less constant state of dilatation, due to some irritation along the track of the nerve fibers in the celiospinal region," and says that they may be found in cases before the evidences of active disease can be discovered. But dilatation of but one pupil is more frequent, some authors saying that it occurs in more than 50 per cent. of cases; that it is an early symptom and may be found before other symptoms and signs make their appearance. It is said to be caused by irritation of the cervical sympathetic by the inflammatory process in the apex and pleura. With the improvement in the disease the difference in the pupils may disappear, but I have seen it persist after the patient recovered. At times, one pupil is unduly contracted.

Muralt<sup>4</sup> pointed out that these unilateral nervous phenomena may be observed within certain limits experimentally after the induction of therapeutic pneumothorax. He found that with the increase in the intrapleural pressure, the pupil dilates and the cheek flushes on the affected side, and in some cases there are typical attacks of migraine, while with the decrease in the pressure the phenomena disappear.

**Pains.**—While a large proportion of tuberculous patients pass through the disease painlessly, there are many who suffer from pains and aches of various degrees of severity. The pains may be in any part of the body, but the most characteristic are those of the chest and upper extremity. Kuthy found that among 650 patients, 60 per cent. had pains in the chest and of these it was localized in 85 per cent. in the affected, or more affected side.

Many of my patients have received the first intimation of trouble

<sup>1</sup> *Gaz. méd. de Paris*, 1869.

<sup>2</sup> *Jour. de méd. et de pharmacol.*, 1894, 241.

<sup>3</sup> *Boston Med. and Surg. Jour.*, 1899, cli, 575.

<sup>4</sup> *Mediz. Klinik*, 1913, ix, 1814 and 1901.



with their lungs through pains which were usually felt in the infra-clavicular space above the second rib and more often in the supra-spinous fossa, between the shoulder-blades or under them. It is usually of a dull character, uninfluenced by motion, breathing or coughing, worse during the night. The skin over the affected area is only rarely tender, but deep pressure almost invariably aggravates it; tapping this region may bring on a coughing spell. Hyperesthesia of the spine between the shoulder-blades is quite common.

In more advanced phthisis pains in the shoulder may be actually agonizing, worse during the night depriving the patient of his sleep and resisting all therapeutic efforts at relief. When occurring in the incipient stage they are not so acutely felt, but may extend all along the arm and forearm down to the finger tips. Minor exposures to the vicissitudes of the weather may bring about pains, and the patient then believes that he is affected with rheumatism. In fact, many cases of "rheumatism" of the shoulder turn out to be phthisis. Diaphragmatic pains are frequent. They are described by the patients as stabbing in character, or as if there was a wound in that region, and are usually due to pleural adhesions and may be aggravated by deep breathing, coughing and sneezing.

Hyperesthesia is very rare in phthisis. The pains are usually elicited by pressure on the regional muscles over the affected parts of the lung. When the apex is affected, the sternocleidomastoidei and the trapezii may be painful; when the lesion is more extensive the scaleni, pectorales and intercostals, and when there is a lesion at the base, the lumbar muscles may be painful on pressure. In pleurisy the same phenomena may be observed. These pains are not due to cough because they are unilateral. They are accompanied by spasmodic contractions of the regional muscles, caused by reflex irritation of the supplying nerves.

These pains have been studied very carefully by Henry Head,<sup>1</sup> James Mackenzie,<sup>2</sup> and more recently in this country by Lovell Langstroth.<sup>3</sup> Head found that these pains were either local or referred, and when due to pleurisy they coincided precisely with the situation of the pleural area involved and were accompanied by deep tenderness, but not by superficial hyperalgesia. In cases of phthisis marked by successive acute or subacute attacks involving previously healthy parts of the lung, referred pains were mostly found. He attributed them to the fact that the end-organs of the sensory nerves in the portion of the lung invaded remained intact and capable of conveying impressions when irritated. These nerve endings were destroyed after the disease advanced, causing necrosis, and were no more capable of causing referred pain. Superficial tenderness is particularly liable to spread along the paths of the nerves and Head believed it due to the cachexia

<sup>1</sup> Brain, 1896, xix, 153.

<sup>2</sup> Symptoms and their Interpretation, London, 1909.

<sup>3</sup> Arch. Intern. Med., 1915, xvi, 149.

and pyrexia characteristic of each acute exacerbation of the disease. Within certain limits, he was able to determine the lung area involved by the cutaneous hyperalgesia. A review of the various forms of pains in phthisis is given by F. Jessen<sup>1</sup> and J. L. Pomeroy<sup>2</sup> in special monographs. It appears, however, that Langstroth's conclusion to the effect that this hyperalgesia is practically of no importance in diagnosis, or in localizing pulmonary lesions, is correct.

It appears that the tenderness found in active phthisis is the result of an attempt on the part of the muscles to protect the diseased viscera beneath them. It is replaced by muscular atrophy in the later stages of phthisis.

The origin of the various pains in phthisis is not always clear. It has been shown by J. Mackenzie that the lung is insensitive to stimulation when healthy or diseased, as is evident from the fact that when an exploring needle penetrates the lung the patient feels no pain. In fact, no form of stimulation of lung tissue seems to be capable of producing sensation, directly or reflexly. It is for this reason that necrosis of lung tissue, as it occurs in gangrene, abscess or tuberculous cavity formation is usually painless.

The suggestion that the pains in phthisis, as well as in pleurisy and pneumonia are due to pleural involvement does not hold either, because the pleura is insensitive. Mackenzie states that he repeatedly explored the pleural cavity for any evidences of sensation and could employ no form of stimulation capable of producing pain. When inducing therapeutic pneumothorax I have repeatedly observed that entering the parietal pleura with the needle produced no pain, nor does scratching the visceral pleura with the point of the needle produce any sensation. Mackenzie is therefore inclined to attribute pains of the kind mentioned above to contraction of the overlying muscles. This is the reason why no hyperesthesia of the skin is met with in phthisis, but pressure pain is frequent. It is due to a visceromotor reflex and occurs along the distribution of the sensory nerves which are stimulated by the lesion. The above-mentioned pain in the shoulder can be explained by irritation in diaphragmatic pleurisy of the phrenic nerve and conducts the stimulus to the skin of the shoulder. Both the phrenic and fourth cervical nerves leave the spinal cord at the same place and the former nerve conducts afferent fibers as well as efferent (motor), and it is in all probability by the former that the stimulus is conveyed to the centre of the fourth cervical nerve in the cord. Pottenger also attributes these shoulder pains to an inflammation of the nerve resulting from the reflex segmental stimulation—a true neuritis. On the other hand, a recent investigation by Capps<sup>3</sup> seems to indicate that irritation of the central part of the diaphragmatic pleura gives referred pain in the neck; and irritation of other parts

<sup>1</sup> Lungenschwindsucht und Nervensystem, Jena, 1905.

<sup>2</sup> Interstate Med. Jour., 1912, xix.

<sup>3</sup> Arch. Intern. Med., 1911, viii.

also give rise to true referred pains, set up by impulses carried to the third and fourth cervical segments by the phrenic nerve, and thence to the areas of these segments.

During the last few days of life the reflexes are usually abolished in the phthysical and they are relieved from all pains; in fact, at times we find them very hopeful because they feel no more pains.

**Psychic Traits.**—Psychoses met with among tuberculous patients may be considered in the main as coincidences, because so many people suffer from phthisis, and inasmuch as this disease is no bar against mental alienation, it is but natural that some should become insane from any of the causes of this aberration. It is a fact that an enormous proportion of insane die from phthisis—Clouston<sup>1</sup> states that two-thirds of deaths among idiots result from tuberculosis—but this may be due to their irrational mode of life, as well as to their confinement in institutions. Delirium is also very often seen in the terminal stages of phthisis and, when not due to meningeal complication, it does not differ from the delirium seen in inanition, exhaustion or febrile intoxication due to other causes. But in addition to these occasional psychic disturbances, which might be expected, there have been noted other psychic disturbances in phthysical patients and many authors have spoken of a characteristic psychology of the consumptive.

Many tuberculous patients show a remarkable change in their mental traits and character, a disturbance in their emotional life and a striking divergence from their previous customs, habits, affections, and tastes. In some, this change precedes the evident onset of the disease, in many it appears synchronously with the symptoms of active disease; it may ameliorate with each improvement and aggravate with each acute exacerbation.

This change in character manifests itself in various other ways: Liberal persons may become stingy and misanthropic, brave ones become cowardly, etc. Engel<sup>2</sup> points out that the original, innate temperament or character of the individual becomes strikingly pronounced in the chronic consumptive: The pessimist suffers from marked despondency; the optimist becomes unreasonably hopeful of the ultimate outcome, etc. These phenomena may be explained by the discordance between the subjective feelings of the patient who is not as disabled as the objective findings of the physician would lead to expect. The mental make up of the patient depends greatly on his physical condition which, in tuberculosis, is subject to great oscillations; aggravations and improvements coming and going quite unexpectedly. The mental traits *per se* do not change, but such traits as were characteristic during youth but, as a result of education, training and the vicissitudes of life, have been suppressed, reappear boldly, unhindered by conventionalities.

A psychic trait of the consumptive which has been noted by most

<sup>1</sup> Allbutt's System of Medicine, viii, 307.

<sup>2</sup> Münch. med. Wehnschr., 1902, xlix, 1383.

writers is selfishness. He becomes egotistical and egocentric. He is interested in the welfare of but one person—himself—to the exclusion of all who have depended on him before. He will eat costly food while his children starve; he will make unreasonable demands on his relatives and friends and show no gratitude. In sanatoriums this has been the most important problem with which the officers have to cope, and the failure of many superintendents is due to their lack of appreciation of this trait of the consumptive. As Saxe<sup>1</sup> states, the ascendance of selfishness plays the most important role in the molding of the mental traits of the tuberculous. In some patients these factors are so pronounced that they completely reveal the concealed elements of their character.

**Euphoria and Euthanasia.**—Optimism, despite many evidences of progressive disease which saps the body, is frequent; only a copious hemorrhage, or, more rarely, a spontaneous pneumothorax, will terrify the average tuberculous patient. Otherwise, all the symptoms amount to little or nothing: An increase in the cough is due to a "cold;" anorexia is caused by bad food, etc.

Barring the functional neuroses, there are no diseases in which suggestion—auto- and heterosuggestion—is so effective in modifying the course of the malady or in relieving symptoms. An injection of water will induce sleep, relieve pain, cough, etc., and even produce an increase in temperature exactly like that of the tuberculin reaction. In many European sanatoriums there is a routine measure before applying tuberculin for diagnostic purposes, to inject water with a view of ascertaining whether the fever is due to psychic effects or to the tuberculin. It has been found that 20 per cent. of patients react to the *injectio vacua*. Some physicians have been able to suggest the hour of the day when the reaction will appear, as well as any or all the symptoms which make up the typical tuberculin reaction. The effects of this high susceptibility to suggestion are seen in phthisiotherapy; quack doctors and remedies are thriving on consumptives more than on any other class of patients, excepting perhaps the venereal in whom the element of secrecy is of importance.

The proverbial euphoria and euthanasia of the consumptive, which have been described in such great detail by many medical authors and which have not escaped the attention of writers of fiction for strong dramatic effects, are other manifestations of the proclivities to auto-suggestion. Experience has taught that when a patient with excessive excavations in the lungs, running high fever, and presenting other symptoms and signs of this condition, begins to believe that he has improved, that he "feels fine," has no pains, does not cough distressingly, we may look for a speedy relief of the unfortunate by that greatest of benefactors for these desperate sufferers, death. It is often astonishing to behold the sinking man make plans for the

<sup>1</sup> New York Med. Jour., 1903, lxxviii, 211 and 263.

future, engage in new enterprises, plan long voyages—not for a cure, which he believes he has almost attained, but for pleasure—or, as I have seen, arranging for his marriage a few days before his death.

Very often this optimism and euphoria are excellent aids in our attempts at curing these patients. It is a well-known fact that there is hardly any hope for a despondent consumptive. On the other hand, this euphoria is occasionally harmful because it misleads the patient and he neglects the instructions of his physician.

It appears that as a result of the prolonged state of intoxication produced by the absorption of the poisons resulting from the metabolism of the tubercle bacilli as well as of the products of decomposition of the affected lung tissue, the consumptive is in about the same mental state as those who are under the influence of mild alcoholic intoxication. The external appearance of the consumptive betrays his state of intoxication. His bright eyes with dilated pupils, which are at times contracted unilaterally, the flushing cheeks, the keen intellect which is so often met with among those who before the onset of the disease were rather dull in this respect, coupled with a flickering intelligence which brightens up suddenly for a few hours but is soon followed by mental depression or fatigue, bear close resemblance to the average person who is under the influence of moderate doses of alcohol or a narcotic drug.

In tuberculous patients, particularly young talented individuals, it is noted that for a few weeks or months now and then they display enormous intellectual capacity of the creative kind. Especially is this to be noted in those who are of the artistic temperament, or who have a talent for imaginative writing. They are in a constant state of nervous irritability, but despite the fact that it hurts their physical condition, they keep on working and produce their best work. This *spes phthisica* has been described by many authors, notably by J. B. Huber<sup>1</sup> and A. C. Jacobson<sup>2</sup> in this country. They maintain that "the quality of genius may, in some cases at least, be affected by tuberculosis," and that the intellectual powers of the genius are quickened by reason of the general psychic exitation resulting from the action of the tuberculous by-products. "They astonish everybody," says Létulle,<sup>3</sup> "with their mental and intellectual activity; their memory, their quick judgment, their delicate reasoning powers are of incomparable amplitude."

The long list of great writers and artists given by Huber and Jacobson, to which many more may be added, shows that tuberculosis is rather frequent among talented individuals, and suggests that it may be enhancing their productivity instead of reducing it as would be expected *a priori*.

**Insomnia.**—Insomnia in the early stages of phthisis may be due to restlessness owing to worry because of the diagnosis of a dangerous

<sup>1</sup> Consumption and Civilization, Philadelphia, 1906.

<sup>2</sup> Interstate Med. Jour., 1914, xxi, 341.

<sup>3</sup> Arch. gén. de méd., 1900, ii, 258.

disease, and is often removed by emphatically reassuring the patient. Indeed, the characteristic attitude of optimism soon prevails and the patient is no more disturbed by insomnia.

In others insomnia is due to excessive cough, or nightsweats, or both. In some cases the administration of hypnotic remedies is of no avail so long as they are given in safe doses. Especially prone to insomnia are patients who suffer from paroxysmal attacks of cough, each fit waking them and keeping them awake for one-half to two hours. In these cases the administration of codein, heroin, etc., is imperative. Profuse nightsweats often act the same way: After waking bathed in perspiration, the patient finds it difficult to fall asleep again.

During the advanced stages many patients find it very hard to sleep because of the copious secretions in the pulmonary cavities which, after a short nap, overflow the bronchi and compel them to rise and expel it from the chest. Some with unilateral lesions may be able to sleep the greater part of the night in certain positions, and they adapt themselves to the conditions. But in others with cavities in both lungs, or with sinuses leading from the cavities in different directions, the prone posture immediately induces cough. Some have to sleep with the face downward if they want to avoid cough, others in the semireclining posture, etc. We also meet with cases in which dyspnea is the cause of insomnia. While during the early stages of phthisis fever may be the cause of insomnia, it is only rarely the case during the advanced stages. The average consumptive has adapted his organism to the fever and does not mind it any more. Tuberculous patients with high fever are often seen sleeping quite soundly as long as the cough, nightsweats, and dyspnea do not disturb them.

In the terminal stage we often observe abnormal somnolence in phthisical patients. For days, at times for weeks, the patient lies in a semicomatous condition, careless about his person, and only now and then wakes to ask for some nourishment. If not due to excessive sedative medication, it may be an indication of meningeal complication. But I have had cases in which this abnormal somnolence has existed for several days or weeks before death, but the autopsy showed no meningeal tuberculosis. Some of these patients have periods when they are mildly delirious.

**Influence of Tuberculosis on the Sexual Sphere.**—The tuberculous toxemia has a profound influence on the sexual organs and their functions. In women, menstrual disturbances are not uncommon during the course of the disease, and quite often these disturbances are noted before the onset of evident symptoms of the disease. In young girls the appearance of menstruation may stay the progress of the disease, as I have seen in several cases. Probably for this reason ancient clinicians thought that amenorrhœa was a cause of phthisis. Now we know it to be an effect of the disease. Amenorrhœa is very frequent during the course of phthisis, and other menstrual disturb-

ances, dysmenorrhea, menorrhagia, metrorrhagia, etc., may be observed in many cases. But I know of a large number of tuberculous women in whom the menstrual function remained practically normal throughout the course of the disease.

During the menstrual days, and at times a few days before the appearance of the flow, there is often observed an aggravation in the pulmonary condition. The fever may rise, the cough increases in intensity, rales increase in number and extent, or reappear in places where they were noted before but had disappeared and new areas of lung tissue are often invaded during this period. Hemoptysis is quite frequent during this period and in rare cases it may even replace menstruation. Premenstrual fever is occasionally noted, as was already stated.

Conception is possible at any stage of the disease, and the pregnancy may, and often does, pass through almost normally, the child being of average weight but of low vitality. Reibmeyr believes that tuberculous women are more prolific than healthy women—nature attempts to compensate in quantity for inferior quality. Abortion and miscarriage are more apt to occur among them than in healthy women. It appears that during pregnancy the tuberculous process is, as a rule, in abeyance and the patient may even improve. Writers of former generations, like Cullen, recommended marriage to tuberculous girls for this reason. Dr. E. Warren<sup>1</sup> in a prize essay published in 1857 said: "Pregnancy, coition, etc., are particularly desired by women affected with phthisis, which constitutes a *pointing of nature* toward a *remedy* for the evils by which the system has been invaded." He quotes the opinions of authorities like Hippocrates, Sydenham, Montgomery, Parr, Rokitansky, Clark and many others, who held similar views on the salutary effects of marriage and pregnancy on tuberculosis. Some modern writers hold similar views. In a paper published in 1897 Charles W. Townsend,<sup>2</sup> speaking of cases observed in the Boston Lying-in Hospital, says that "during pregnancy the patient often seems better and the disease appears in abeyance," and that "nature seems to put forth a supreme effort to suppress the disease during pregnancy and to make the labor easy and short, but after the child is born the disease advances at a rapid rate."

There is no question that during pregnancy the more annoying symptoms are in abeyance in many cases. In fact, it is rare to see a woman becoming sick with progressive disease during the period of pregnancy. But after the child is born the disease flares up and often begins to progress with frightful rapidity. A considerable proportion of tuberculous women date back the beginning of the disease to childbirth. Labor seems to stimulate the process in the lungs and favors the development of progressive disease. Women in the incipient stage of phthisis, and those in whom the disease was arrested or even

<sup>1</sup> Amer. Jour. Med. Sci., 1857, xxxiv, 87.

<sup>2</sup> Boston Med. and Surg. Jour., 1897, lxxxviii, 391.

cured, are apt to suffer an extension of the process, or a relapse or recurrence of active phthisis after pregnancy and childbirth.

**Sexual Irritability.**—The popular views entertained by the laity and the profession to the effect that consumptives have excessive sexual potency and demands are apparently well founded. Even during the incipient stage of the disease there is often noted an increased sexual irritability, and this is apparently the reason why some believe that phthisis is at times due to excessive venery. Létulle asserts that sexual excesses are common at the commencement of the disease and are checked only when the limit of exhaustion is attained. W. H. Peters<sup>1</sup> observed a tendency to abnormal sexual excitement so frequent among consumptives as to require the careful attention of the physician. He also says that "every physician has been impressed by the almost disgusting, and sometimes revolting persistence of the sexual instinct in consumptives, even late in the disease."

It is noteworthy that in the advanced stages of the disease, when the body is extremely emaciated, the muscles atrophied and the vital forces apparently at their lowest, sexual potency may be retained. Even shortly before his death a consumptive may impregnate his wife, and a woman who has lost half her normal weight and is subject to frequent hemorrhages, runs a febrile temperature, sweats and coughs distressingly, is at times seen in a pregnant state. Peters quotes H. L. Barnes, superintendent of the Rhode Island Sanatorium, about a patient who died from a hemorrhage coming during the sexual act which took place while on a visit from the sanatorium to his wife. I have seen several somewhat similar cases. In hospitals for advanced consumptives the patients must be watched in this regard, especially when the male division is not completely separated from the female division. Sexual excesses, according to Gimbert,<sup>2</sup> often hasten the fatal outcome of the disease.

Other writers deny altogether that consumptives are more sensuous than others. Karl von Ruck,<sup>3</sup> in a review of the subject, arrives at the conclusion that "phthisis is not a cause of sexual excesses, there being no difference between tuberculous and non-tuberculous subjects; that in the advancing disease the sexual functions decline the same as they do in other wasting diseases." But the bulk of the evidence appears to favor the view that excesses are more common among consumptives than among others.

These sexual excesses have been attributed to the tuberculous toxemia, but others have denied this explanation. It has been stated that the lazy, indolent life, the lack of muscular exercise, and the excessive consumption of nitrogenous food during the treatment are more responsible for the sexual proclivities than the tuberculous toxemia. It has also been stated that in sanatoriums the association

<sup>1</sup> Jour. Amer. Med. Assn., 1908, 1, 938.

<sup>2</sup> Rêvue de la Tuberculose, 1907, iv, 1.

<sup>3</sup> Amer. Jour. Dermatology, 1907, xi, 284.



of the sexes favors tendencies in this direction. In many the despondency engendered by the knowledge of suffering from an incurable disease urges the patient to take in as much of life and its pleasures as possible before it is too late.

There are other chronic diseases in which the patients are idle, eat well and may be despondent, yet they do not indulge in sexual excesses to the same extent as the tuberculous, which would be in line with the suggestion that the tuberculous toxemia is effective in the direction of causing sexual irritability. Turban found that in artificial tuberculin poisoning, *i. e.*, when tuberculin is administered for therapeutic purposes, sexual irritability is increased, and in some cases he had to discard specific treatment for this reason. "Every physician with a large experience with tuberculous patients," says Muralt, "knows of cases in which recovery from the disease brought about normal functions in this regard."

Weygandt<sup>1</sup> made a collective investigation of this problem among physicians in German sanatoriums in which incipient cases are admitted. Many of the answers were to the effect that they had not observed any special increase in the sexual desires of their patients; three directors of sanatoriums, Kohler, Krause, and Marquard, sent the interesting information that the patients had accused the doctors of secretly putting aphrodisiac or anaphrodisiac drugs into the milk or other food. It appears that in many German sanatoriums such superstitions prevail, thus indicating that the patients themselves are aware of the increased sexual irritability.

<sup>1</sup> Mediz. Klinik, 1912, viii, 91 and 137.

## CHAPTER XIV.

### INSPECTION AND PALPATION.

**The Stigmata of Phthisis.**—After the history and symptomatology of the patient have been carefully inquired into, the physical examination should begin with inspection of the physical make-up of the individual. In phthisis not only the chest should be carefully examined but also the head, the face, the neck, the abdomen, and the extremities. The stigmata of this disease are often scattered over various parts of the body, and the experienced eye may, at times, find outside of the region of the chest certain signs which are highly suggestive of phthisis. In some borderland cases these stigmata may be of great assistance in formulating an opinion on the diagnosis and prognosis.

**Complexion.**—Hippocrates described the *habitus phthisicus*—the “form of the body peculiarly subject to phthisical complaints”—as characterized by a smooth, whitish skin, blue eyes, blond or reddish hair, and a phlegmatic temperament. Following this ancient clinician, many modern writers on this subject have stated that the external appearance of certain persons betrays a strong predisposition to this disease.

Hippocrates's notion that blond-haired and blue-eyed persons are more prone to phthisis has survived to this very day, and Beddoe, Landouzy, Delpeuch, Piéry, Woodruff, and many others hold the same view. Exact information, however, does not sustain this opinion that fair-complexioned people are more prone to tuberculosis. In countries with predominant blond populations, like Scandinavia, England, Northern Germany, etc., the consumptives are generally blonds; while in Italy, Spain, Greece, etc., where the dominant racial elements are brunettes, the consumptives are of the same complexion, as can be seen on visiting the sanatoriums in these countries. In China and Japan there are no blonds, yet tuberculosis is not lacking. Evidently infection, the length of time a people has been exposed to the tubercle bacilli and, above all, social and economic conditions are of greater importance in determining the rates of morbidity and mortality than race or color.

**Facies.**—The confirmed consumptive presents a characteristic, in fact, an unmistakable appearance, which betrays his disease not only to the experienced physician, but also to the laity, and he can often be picked out from a group of healthy people with comparative ease and certainty. The emaciated body, the pallor of the face with the hectic flush on the cheeks, the round shoulders and the bodily decrepitude may be seen in other wasting diseases; but the facies of the

consumptive, while possessing all these traits, has other characteristic stigmata. In very few other diseases is there to be seen such a typical facial expression as in the consumptive.

The facial muscles are wasted, the cheeks sunken and the malar bones protrude; the lips are pale or livid, often contracted as if smiling or grinning; the hectic flush, which may be unilateral; the thin neck appears longer than normal, the sternomastoids are accentuated like two tense bands on both sides; the head is bent forward between the two round shoulders and the spine is bent. Because of the wasting, the ears appear larger; one may be redder than the other.

But the most pathognomonic parts of the cast of countenance of the consumptive are his eyes. They are deeply set in the sockets, which are larger than normal because of the wasting of the orbicularis palpebrarum. We also meet with a widening of the palpebral aperture and a slight protrusion of the eyeball on the affected side as a result of irritation of the sympathetic. A narrow palpebral aperture with a somewhat deeply set eyeball, is a symptom of prolonged irritation of the nerve paths, and is met with in cases with adherent apical pleura, as was shown by Kuthy. To the same cause has been attributed unilateral dilatation, or more rarely, contraction of the pupil which may precede the evident onset of active disease.

The appearance of the eye as a whole is pathognomonic and can be more easily recognized than described. It has a characteristic brilliancy which has been described as transparent, lustrous, bright, dimly brilliant; it differs from the brilliancy of the eyes in other fevers in the fact that it appears gloomy, dismal or haunted—its glance can always be felt. Some have attempted to explain these characteristics as due to the widely dilated pupils, while the pearly-white sclerotics are said to be an expression of vasomotor succulence of the bulbar conjunctiva resulting from pressure on the cervical sympathetics and are to be seen mostly in cases of adherent apical pleurisy.

This facies has been recognized by the laity, and the folk-lore of Europe abounds in sayings about the facial expression of the consumptive. Writers of fiction and painters have also considered it "interesting" and make great use of it in their productions. Many of the classical and modern painters have depicted this cast of countenance showing the false euphoria of the smiling, tranquilly bright, yet melancholy eyes of the consumptive, which are perhaps best seen in Leonardo da Vinci's *La Gioconda*—a picture of a phthisical face superior to any description that can be given of it.

I have seen these facies in some patients with latent or quiescent tuberculosis in whom physical exploration of the chest showed but indefinite signs of a lesion. It appears to be especially marked in persons of phthisical stock; in other words, those who were infected during childhood, but have more or less recovered.

**The Skin.**—Other stigmata of phthisis, which may be noted in the early stages of the disease, should be mentioned. On the forehead and

upper parts of the cheeks we may see cholasma phthisicorum, and, in those who sweat profusely, pityriasis versicolor and tabescentium on the anterior and posterior aspects of the chest. In those who suffer from dyspnea we may find clubbed fingers or deformities of the hands, wrists, spine and ankles, which are the results of pulmonary osteoarthropathy. On the neck, spasm or atrophy of the muscles, which will soon be described, may give us a clue that a careful examination of the chest is indicated.

**Enlarged Glands.**—Visibly enlarged glands are quite rare in adults, though I have seen two cases in which they went on to suppuration. But palpable glands on the neck are very frequent—in at least 50 per cent. of my cases. In children, enlarged glands are very frequent, but they are not always an indication of tuberculosis. If enlarged cervical glands were pathognomonic of tuberculosis in children we should find very few who live in poverty free from this disease (see Chapter XXIV). Of greater importance from the diagnostic standpoint is enlargement of the supraclavicular glands, especially when found unilaterally, and it speaks for tuberculosis of the costal pleura.

We also very often find enlargement of the thyroid gland in tuberculous subjects, at times in the incipient stage. The reciprocal relation between hyperthyroidism and tuberculosis is a mooted question.

**Enlarged Veins on the Chest.**—Enlarged veins are often seen on the chest, especially in the infraclavicular region over the first and second interspace and posteriorly opposite the first thoracic spine, and below along the line of insertion of the diaphragm. The upper enlarged veins are caused by the interference with the emptying of the internal mammary and intercostal veins because of pressure on the vena azygos by swollen thoracic glands, and also by the increased expiratory efforts while coughing. They are occasionally seen in healthy persons, especially in nursing women, and they may be unilateral in patients suffering from chronic bronchitis and pulmonary emphysema, as well as with endothoracic tumors. According to Lombardi,<sup>1</sup> the varicosities in the neighborhood of the seventh cervical and first thoracic vertebrae may be seen in 80 to 90 per cent. of cases of phthisis, but I see them very frequently in persons without any active pulmonary disease.

It will also be noted in some cases that the nipple is lower or located more externally, while in women the mammary gland may be smaller and the nipple may be less pigmented than on the opposite unaffected side.

**The Phthisical Chest.**—Hippocrates, Galen, Aretæus and other ancient clinicians mentioned the phthisical chest, and modern text-books devote considerable space to giving details about its form, shape and significance, notwithstanding the fact that many persons with "phthisical chests" pass through life unscathed, while many consump-

<sup>1</sup> *Gior. internaz. di Scien. med.*, 1913, xxxv, 751.

tives have at the beginning of the disease excellent chests. There was a time when everyone who had a deformed chest, especially of the type called flat, was considered tuberculous or, at least, predisposed to the disease. By actual measurement, Woods Hutchinson<sup>1</sup> found that the chest of the consumptive is altogether unusually round, the sternodorsal diameter is comparatively large in the consumptive when compared with the average healthy person, and he suggests that it is due to a persistence of the infantile thorax in the adult. These observations have been confirmed by Bessesen,<sup>2</sup> Niles and others.

The problem whether the phthysical chest is a cause, congenital or acquired, of tuberculosis, has also been raised. As will be shown later, all evidence tends to show that it is an expression of intrathoracic disease and thus a result of tuberculosis during childhood.

**The Normal Thorax.**—Before looking for the pathological chest we must have a clear idea as to what constitutes a normal thorax, and it should be stated at the onset that a well-formed thorax is an ideal which cannot be encountered more often than a perfectly normal physique in the individual. I can do no better than quote Pottenger's<sup>3</sup> description, which is as complete and thorough as can be given:

“Such a thorax in an adult should be symmetrical on both sides. Beginning at the clavicle it should bulge forward, reaching the maximum point on a level with the third or fourth rib and then gradually flatten out again as the lower border of the ribs is reached. The supraclavicular and infraclavicular spaces should be well filled and almost even with the clavicles themselves. The scapulæ should stand symmetrically; the ribs and intercostal spaces should be well covered with subcutaneous tissue and muscles so that the intercostal spaces are barely recognizable in the upper two-thirds of the thorax, and are only seen distinctly in the lower portion where the musculature is thin. There should be a general symmetry in the muscles of the two sides, no individual or group of muscles standing out with undue prominence unless it be those that are increased in size by greater use, such as the deltoides, trapezius, rhomboides and pectorales in persons who do heavy work and use one hand more than the other. The anterior neck muscles should not stand out unduly, unless the patient is emaciated. Neither should the neck and chest muscles appear degenerated or atrophied under normal conditions.”

While such an ideal chest is only rarely seen in healthy persons, it is never seen in a consumptive. In the latter, going hand-in-hand with the progress of the disease, the form and shape of the thorax changes, as a result of certain changes in the respiratory muscles, and in many cases we find on inspection and palpation conditions which are characteristic of the phthysical chest.

<sup>1</sup> Jour. Amer. Med. Assn., 1903, xl, 1196.

<sup>2</sup> *Ibid.*, 1905, xlv, 2003.

<sup>3</sup> Muscle Spasm and Degeneration in Intrathoracic Inflammations, St. Louis, 1912, p. 15.

**Technic of Inspection and Palpation of the Chest.**—In addition to the light, warm room and stripping the patient to the waist, which are self-evident requirements, the patient is to be seated on a round stool, directly facing the window or the source of artificial light. He is permitted to assume his natural posture without urging him to sit straight up, hold his head in the middle line, etc., so that we may note any faulty position of the head, neck, spine and chest. Careful attention is to be paid to the position of the head, the shoulders, the clavicles, the ribs and the scapulæ during rest and during moderate and forced breathing.

Above all, we are looking for evidences of asymmetry in structure, form and mobility, when the two sides of the chest are compared. Motion can be ascertained by inspection, carefully noting from a distance the tips of the acromion processes, as well as the elevation of the ribs during inspiration, the position of the scapulæ during both phases of the respiratory act, and also the lateral expansion of the lower parts of the thorax. Flattening, excavations and undue prominence of the respiratory muscles are to be especially looked for. The supraspinous and supraclavicular fossæ are compared and any deviation from the normal should not be overlooked. Spinal deformity, if present, must be given attention because it may be the result of an intrathoracic lesion, and also because it may have an immense influence on the results obtained by percussion and auscultation, and also on the skiagram.

The motion of the anterior aspect of the thorax is best studied while standing behind the patient and looking over his head watching the ribs and clavicles as they rise and descend during inspiration and expiration, and noting any retardation or limitation of motion on one side as compared with the other. It is, however, best to ascertain this by palpation, placing the hands on each side of the patient's neck, the thumbs meeting behind at the spine and fingers reaching down over the clavicles (Fig. 39), and for the lower parts by placing the hands over the lateral aspects of the chest. In this manner slight differences can be detected more easily than by inspection. Special attention is to be paid to lagging—one side of the chest is delayed in movement and, in more advanced cases, expansion is limited. At times we meet with both lagging and limitation of motion in various parts of the chest and we may conclude that the former is an indication of a recent lesion, while the latter is caused by an old, probably pleuritic lesion.

Spasm and degeneration of muscles of the neck and chest are best ascertained by Pottenger's method of "light touch palpation." Pressing the tips of the fingers over the muscles under consideration and moving the hand sidewise, carefully noting the degree of resistance, will show this condition. While doing this the fingers should not be allowed to slip on the skin because it is the condition of the muscles and not of the skin that we wish to ascertain. Over acute lesions it

is found that the muscles give to the palpating fingers a distinct feeling of increased resistance, that they are firmer and fuller than normal, while over advanced lesions there is a flabby, doughy feeling and the bundles can be easily separated owing to atrophy and degeneration.

**Significance of Lagging.**—In the very incipency of a pulmonary lesion we often note that the affected side of the chest begins to expand and the shoulder to move upward later than the opposite healthy side of the chest, and finally does not attain the same amount of expansion. In far-advanced cases there may even be absolute immobility of the affected side. It is best ascertained by letting the patient first breathe normally and then asking him to take a few deep inspirations.

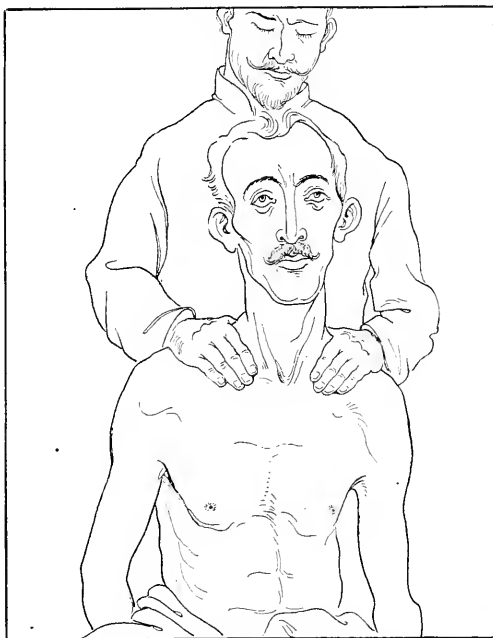


FIG. 39.—Testing mobility of the chest.

Lagging of the upper part of one side of the chest is an indication of a lesion in that apex, provided an acute or chronic non-tuberculous inflammatory process of the lung and pleura is excluded. When the motions of both sides are equal, but there are sure signs of tuberculosis, we may conclude that there is a bilateral lesion. With an old quiescent lesion in one side and a new and active lesion in the other, the lagging is more pronounced in the newly affected side. I often find difficulties in clearing up by inspection and palpation old bilateral lesions in which both sides show limited motion. In these percussion and auscultation give more reliable information. But in incipient unilateral cases inspection is of immense value.

**Thoracic Asymmetry.**—Looking at the phthisical chest anteriorly, in cases in which the disease has already made some inroads, we find some undue prominence, even arching of the clavicle and more or less deep excavation in the supra- and infraclavicular fossæ, more marked or exclusively on the affected side. The angle of Louis at the junction of the manubrium and the gladiolus is more pronounced than in the average healthy chest. Posteriorly, we find kyphosis in many cases, the scapulæ are prominent, winged and even dislocated, nearer the spine on the affected side. The intercostal spaces are rather wide and deep and in extreme cases the free margins of the costal cartilages nearly meet in the middle line. In addition to these changes we meet with distortions of various parts of the chest, especially the upper half—flattening and retractions of various degrees anteriorly and

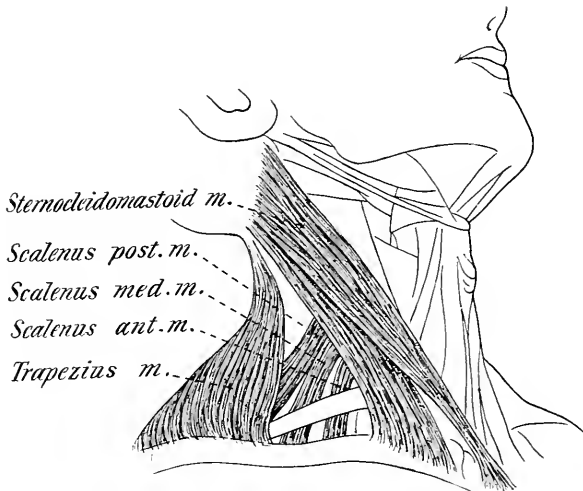


FIG. 40.—Muscles of the neck which are either spasmodically contracted or atrophied in pulmonary tuberculosis.

posteriorly. Depression of the acromial end of the clavicle on the affected side may be already noted in the very early stages of the disease. Kuthy<sup>1</sup> found it in 82 per cent. of his incipient cases.

**Spasm and Degeneration of the Thoracic Muscles.**—Any or most of these changes in the contour of the chest may be noted in cases of non-tuberculous affections of the thoracic viscera, and also in patients who had a tuberculous lesion which had healed, the patient being in excellent health. Pottenger, in his epoch-making studies of the tuberculous chest, has given us certain clues as to the means of differentiating these conditions. It appears that intrathoracic conditions have a great influence on the muscles of respiration, a fact which

<sup>1</sup> Sixth Internat. Congr. Tuberc., 1908, i, 1215.



has been known for a long time, but was only rationally interpreted and made available for rational diagnosis by Pottenger.

Whenever the lung or pleura is acutely inflamed, the thoracic muscles over the seat of the lesion are in a state of spasmodic contraction, like the abdominal muscles in a case of appendicitis. Depending on the acuteness of the inflammatory process in the pulmonary parenchyma or pleura, the muscles of the neck and chest show this contraction in various degrees.

Inspection and palpation reveal this condition very clearly in the vast majority of cases. Muscles in spasm are larger and firmer in appearance as well as to touch, giving a distinct feeling of increased tension. Often the more tendinous parts of muscles feel like distinct cords, while the more fleshy parts are larger and firmer to the touch than normal muscles on the opposite unaffected side.

After the inflammatory process in the lung and pleura has lasted for some time and passes into a chronic stage, the muscles degenerate; they waste and become flabby. To the palpating finger they feel doughy, their normal tone or elasticity is gone, and their bundles are easily separated. It is important to note that coincident with this change in the muscles, there is always seen atrophy of the skin and a disappearance of the subcutaneous tissue. Some of these changes are evident to the sight as well as to the touch.

Pottenger looks upon these muscle changes as due to reflex stimulation of the motor nerves, the result of continuous irritation caused by the impulse from the inflamed lung and pleura. When this irritation is kept up very long degeneration and wasting follow, though the latter may be due partly to trophic disturbances. But if it is true that we can make out by superficial palpation of the dead body internal solid structures it would indicate that the theory of reflex irritation is inadequate.

**Muscular Changes in Incipient Cases.**—In incipient cases we often find that the sternocleidomastoid, the scaleni and pectoralis anteriorly and trapezius, levator anguli scapuli, etc., posteriorly, are in a state of spasm: They stand out more prominently, are larger and firmer to the touch than the same muscles on the opposite, unaffected side. I have often seen that as a result of this spasm the supraspinous fossa was fuller at first sight. When occupational influences can be excluded, it is a good sign of active incipient phthisis. When combined with lagging of the same region or at the base of the same side, it is undoubtedly a sign of a lesion of the lung, provided non-tuberculous disease can be excluded.

**Muscular Changes in Advanced Disease.**—With the advance of the disease, the affected muscles, as a result of prolonged spasm, begin to atrophy and degenerate. The result is that on inspection and palpation even better criteria of the intrathoracic condition may be elicited. The degeneration of the skin and subcutaneous tissue over the site of the lesion is seen at once; the skin can be lifted up

with the fingers more easily and it is felt that it lacks the normal elasticity. The sternocleidomastoid, scaleni, pectoralis, trapezius, levator anguli scapulæ and rhomboidei all look smaller than their mates on the unaffected side. They are flabby and doughy to the touch.

In cases with old circumscribed lesions limited to the upper part of the apex we may find the upper half of the pectoralis degenerated

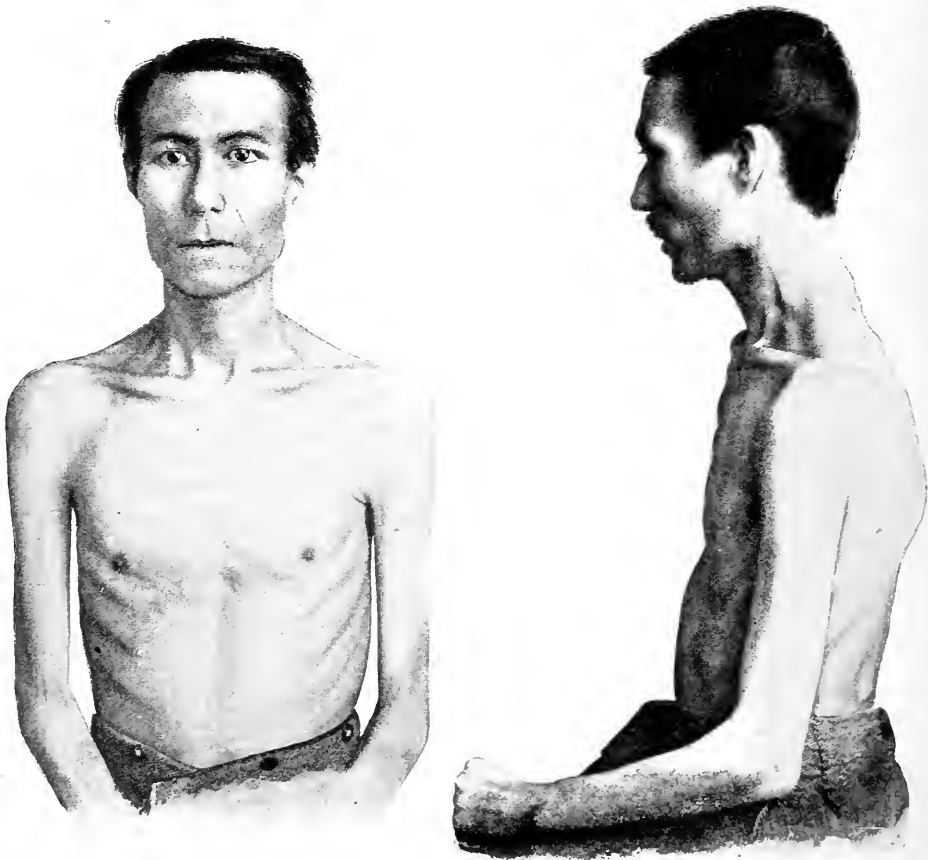


FIG. 41.—The phthisical chest. Full-blooded Indian. (Musser.)

and flabby, while the lower half is normal. As a result of atrophy of the trapezius we find flattening of the supraspinous fossa; in extreme cases it appears cupped. In old cases extension of the disease may often be ascertained by inspection and palpation. The old lesion on one side shows wasting of the skin and muscles, while on the opposite side, where tubercles have just caused a new incipient lesion, the muscles are in spasm—contracted and prominent. Lagging is more

pronounced on the newly affected side; it indicates an active lesion which hinders motion of the contracted muscles, especially the diaphragm. "When palpation, percussion and auscultation show evidences of a lesion and there are changes in the mobility of the suspected side and no spasm of the muscles over the apex but, on the contrary, the tone of the overlying muscles has decreased and there are evidences

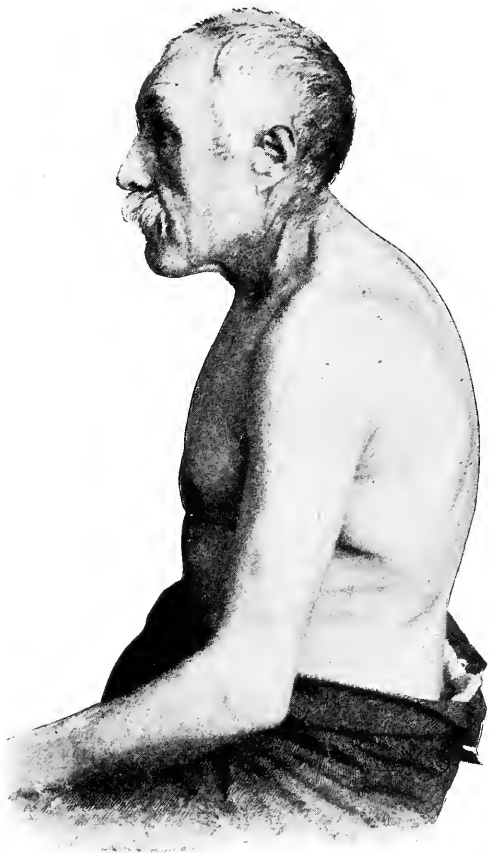


FIG. 42.—Emphysema with enlargement of the chest; the anteroposterior diameter is much increased. (Musser.)

of atrophy of the subcutaneous tissue combined with clinical symptoms of tuberculosis, we are justified in concluding that we deal with an old, inactive or healed process." (Pottenger.)

In many cases we may find the regional muscles more or less atrophied from disuse, especially when compared with the opposite side where they are enlarged, firm and prominent because of excessive occupational hypertrophy. This is best differentiated by bearing in

mind that in muscular atrophy due to disuse, the subcutaneous tissue is normal, while when due to a pulmonary lesion it is atrophied.

**Effects of Muscular Atrophy on the Thorax.**—The lagging which was formerly attributed to lack of expansion of the affected lung or to pleural adhesions, is better explained by the tonic contraction of the scaleni and sternocleidomastoid on the affected side which raise and fix the sternum and immobilize to a certain extent the first and second ribs, thus limiting the respiratory motion of the affected side. Round shoulders, which were formerly attributed to weakness of the posterior muscles which hold the spine erect, are more rationally explained by Pottenger as due in a great measure to shortening of the anterior muscles through spasm and degeneration, together with lessened mobility of the thorax. Flattening of the chest, especially over pulmonary cavities, which was formerly attributed to atmospheric pressure forcing the bony thorax to contract in order to occupy space previously occupied by lung tissue, is explained by Pottenger as due to inflammatory disease within the thoracic cavity and reflex interference with the normal motion of the diaphragm which is known to be part and parcel of phthisis from radiographic studies.

Bearing in mind that most are infected with tuberculosis during childhood but that the pulmonary lesion heals or remains latent, it is understood that the lesions produce muscular changes in the manner described above during the time of their activity. Thus, we have an explanation for the origin of the phthisical or paralytic thorax. It is a result of an earlier infection which has healed and is not a predisposing cause of phthisis. A careful study of children of tuberculous parentage has shown that they are born with normal chests, and the characteristic deformity only occurs later in life after they are infected with tubercle.

Palpation for the vocal fremitus is of no diagnostic value in any stage of phthisis, excepting in cases where pleural effusions are suspected. But it is often absent in thickened pleura and thus is not of assistance in our attempts at differentiating the latter from an effusion.

## CHAPTER XV

### PERCUSSION OF THE CHEST IN PHTHISIS.

WHILE the value of percussion in the diagnosis of conditions in the advanced stages of phthisis and its complications is not questioned, it has been very seriously debated whether it can give dependable information in the early or incipient stage. Many authorities, notably of the French school, like Grancher, Bezançon, Barbier, Piéry; and also S. West, Bonney, Lawrason Brown, Henry Sewall and others maintain that small tuberculous foci in the lung in incipient phthisis can be recognized solely through recourse to auscultation, and that when dulness is elicited on percussion we may be confident that we are dealing with extensive infiltration—a more or less advanced stage of the disease. On the other hand, Aufrecht, Krönig, Goldscheider, Ewart, Lees, Riviere, and many others maintain that if we are to detect incipient lesions in phthisis, we must resort to percussion and it is only when the process has advanced that definite auscultatory signs are elicited.

**Aims of Percussion.**—It seems that these differences of opinion are mainly due to a misapprehension as to the aims of percussion. Those who expect to make a diagnosis relying solely on percussion findings will be sadly disappointed, just as they will fail in attempting to draw final conclusions from any other single symptom or sign. Percussion only gives information about the density or the air content of the lung at the point examined. Whether an airless area thus detected is due to a tuberculous infiltration, or to one of the numerous other factors that may consolidate large or small areas of lung tissue, must be determined by a study of all the concomitant symptoms and signs. On the other hand, given symptoms of phthisis such as cough, fever, anorexia, etc., signs of a limited infiltration elicited on percussion may enable us to localize the process and complete the diagnosis in the absence of auscultatory signs.

We must bear in mind that phthisis does not begin as a catarrh of the small bronchi, as some believe, but as an infiltration, transforming the normal porous, air-containing and resonant lung into solid non-resonant tissue. At this stage the alveoli are filled with exudate, or the interstitial tissues contract and compress the alveoli, finally obliterating them altogether. Inasmuch as altered breath sounds and rales can only be found in the pulmonary apices when edema and secretions interfere with the entry or exit of the air current while passing through the air vesicles and bronchioles, it is clear that auscul-

tation may not give any information at a very early stage. As long as the infiltration remains beneath the mucous membrane of the bronchi, the entrance of air into the alveoli of the affected area is not interfered with very much, while in the rest of the lung it is freely circulating. Auscultation may not reveal such a lesion which is surrounded by healthy lung tissue working vicariously and sucking in more air.

It is only when the caseous material of the infiltrate softens and breaks through the wall of a bronchus, thus permitting the entrance

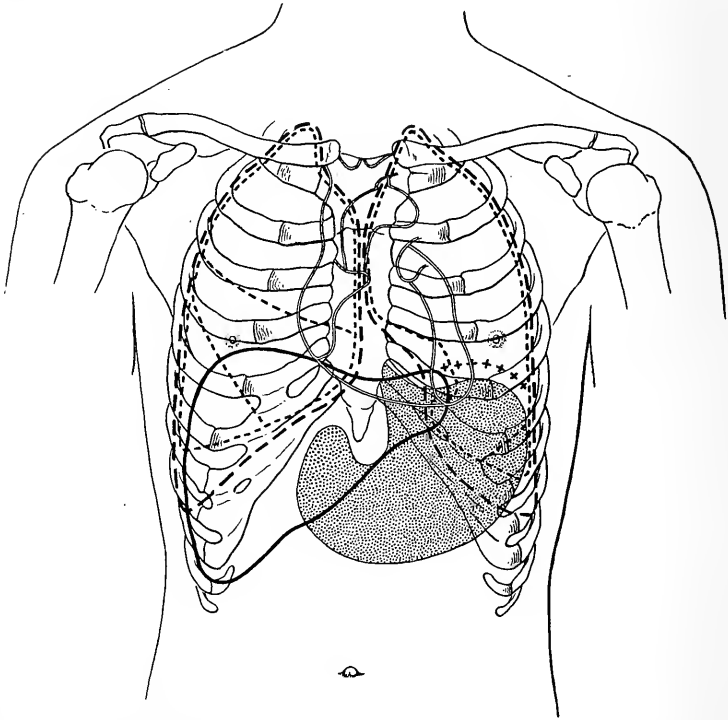


FIG. 43.—Outlines of viscera. The margins of the lobes of the lungs are shown (interrupted line - - - -); solid black line, heart, liver, and spleen; stomach shaded. (After His-Spaltenholz, Luschka, and Musser.)

of air into the disease focus proper that rales can be heard on auscultation. At that time tubercle bacilli make their appearance in the sputum. When we have rales we may be sure that we are dealing with a more or less advanced stage of the disease—caseation and softening have already taken place.

When the tuberculous process is not located originally in the bronchioles, but in the peribronchial tissues, it is again evident that the air circulating in the bronchial tree cannot reach the tubercle at all, and the auscultatory signs will necessarily be negative. At most, feeble

or absent breath sounds over a limited area may be the first sign elicited.

**Technic of Percussion.**—Percussion has been neglected by many because it has not given them the information they sought; at times it even misinformed them. The reason is almost invariably faulty technic. Before giving details as to percussion findings in early phthisis, we must speak about the proper technic to be followed in apical percussion.

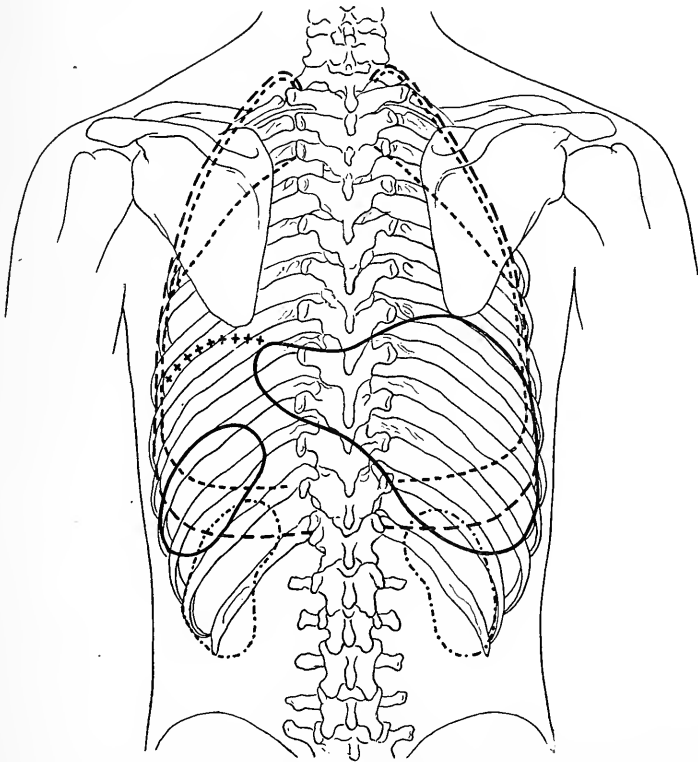


FIG. 44.—Outlines of viscera. The margins of the lobes of the lungs are shown (interrupted line - - - - -); solid black line, heart, liver, and spleen; stomach shaded. (After His-Spaltenholz, Luschka, and Musser.)

The first and most important point is a light stroke with the finger. Heavy blows with two or three fingers are worse than useless. Because of the elasticity of the thoracic walls, a great part of the percussion stroke is always dissipated along the muscular and bony parieties, and when we strike a heavy blow most of the force is conducted laterally by the ribs and intercostal muscles, which are set into strong vibration, acting as large pleximeters, and resonance from all the lung beneath them is elicited. Small areas of airless tissue are thus overlooked. With a light stroke the force is not conducted along the parieties, but

penetrates sagittally into the lung, affording information about its condition immediately beneath the point examined.

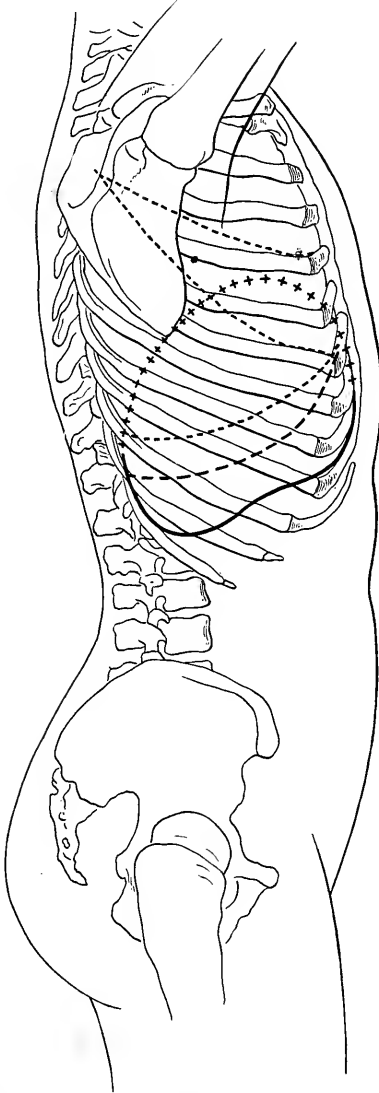


FIG. 45

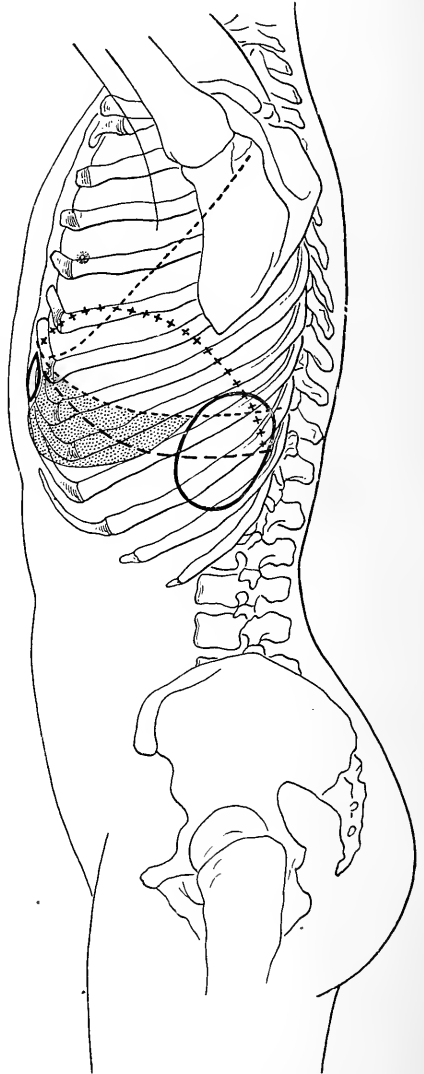


FIG. 46

FIGS. 45 and 46.—Margins of the lungs and of individual lobes, dotted line (-----); limits of pleural sacs, interrupted line (-----); liver and spleen, solid black line; diaphragm, starred line (\*\*\*\*\*); stomach (portion not covered by lung) shaded. (After Luschka and Musser.)

With light percussion in which the stroke is gentle and soft, hardly audible at any distance, we can always localize areas of superficial



dulness. Deep-seated, airless areas cannot be detected by heavy percussion, as is evident from the fact that we cannot map out the heart from behind, and in obese and edematous persons it is quite difficult, often impossible, to define the boundary between the liver and the lung. Strong blows do not reach much deeper into the pulmonary tissue proper than light strokes. To be sure, they set up stronger vibrations, but mainly in a lateral direction and for this reason the penetrating power of the heavy blow may be even less than that of the light stroke.

Gentle percussion often brings out small areas of dulness which disappear with an increase in the force of the blow because larger areas have been set into vibration. This point is utilized for diagnostic purposes: If, on increasing the force of the blow, the dulness remains, we may be sure that we are dealing with extensive areas of airless tissue.

**The Pleximeter Finger.**—Light percussion is best accomplished when the movement of the percussing finger is exerted only from the metacarpophalangeal joint. The note elicited should be only a faint sound which can be heard when listening attentively. Of course perfect silence must be maintained in the room. When reaching an airless area, the contrast between the resonance evoked in the air-containing space and the deadness over the dull area is striking. It has been well said that the contrast between something and nothing is easier of appreciation than the difference between one thing and another which differs but slightly from it. Over resonant areas we evoke a note, while over dull areas no note is brought out at all.

Strong pressure of the pleximeter finger on the chest wall dissipates the advantages of light percussion by bringing the intercostal muscles into tension, making them large pleximeters, which elicits resonance of the neighboring air-containing lung, and small areas of dulness can thus not be delineated. Very light contact of the pleximeter finger with the chest wall is therefore important; in delicate percussion, the mere weight of the finger is sufficient, as Sahli points out.

Bearing in mind that, as a rule, tuberculous lesions spread from above downward and that the line between the healthy and infiltrated tissue usually runs horizontally, we must percuss from above downward or the reverse in horizontal zones. The pleximeter finger should be placed parallel with the ribs (Fig. 47) and not perpendicular to them as is often done. It is obvious that when the pleximeter finger is placed vertically on the chest we obtain mixed resonance because the stroke brings both healthy and diseased lung into vibration in cases of limited lesions. Only intercostal spaces should be percussed because percussion of the ribs, which in themselves are to be considered as long pleximeters, brings out resonance due to vibrations of large areas of lung tissue which lie laterally and not only from beneath the spot which we intend to strike at the given moment.

The usual way of beginning percussion at the top of the chest and

going gradually downward to the base has many disadvantages. It is much better to percuss from below upward. N. K. Wood<sup>1</sup> summarizes the reasons for this procedure as follows: "It is much easier for the ear to pick up a higher note from a lower than it is to do the reverse; it requires a much lighter stroke to bring out the normal note than the pathological; it is the rational plan to work from the normal as a standard toward the pathological. The reverse leads to faulty standards. The apices, as is well known, are most frequently affected and more rarely give a normal note. To start at the apex, therefore, is usually to commence with a pathological note. This

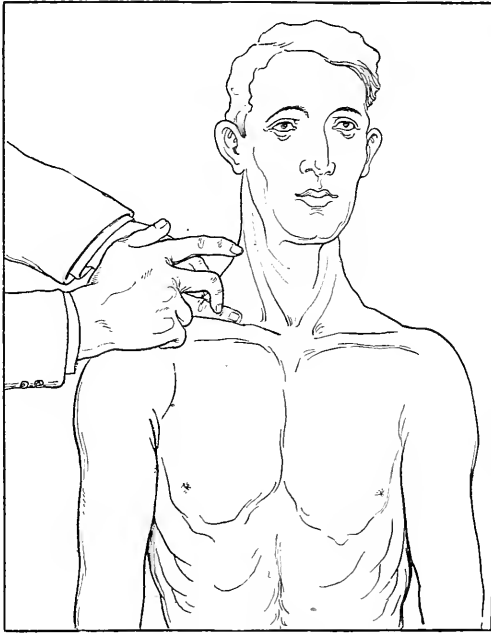


FIG. 47.—Percussion of the right apex.

prejudices the further examination. With downward percussion, the higher note emerges into the lower too imperceptibly to do accurate work. This is so for two reasons: (1) the mind becomes prejudiced in favor of a pathological note and consequently does not attempt to make fine distinctions, (2) a heavier stroke is required for the pathological note and when the more resonant is reached, the percussion is continued too heavily to detect what should be readily appreciated differences in the force of stroke necessary to bring out a good note. In this way the examiner deprives himself of a very important guide to collect accurate data."

<sup>1</sup> Jour. Amer. Med. Assn., 1914, lxiii, 1378.

**The Hooked-finger Pleximeter.**—In incipient phthisis we aim at localizing the smallest possible area of dullness, and at times the pleximeter finger is too large for the purpose. Plesch<sup>1</sup> has suggested that the pleximeter finger be flexed at the second phalanx to a right angle, the pulp is only applied to the chest and distal end of the first phalanx is percussed (Fig. 48). This manœuvre also enables the delimitation of the boundaries of the apex, or the determination of the condition of the apex behind the heads of the sternocleidomastoid, which is often of great importance.

**Position of the Patient.**—The patient should sit on a revolving stool, or better stand up with his head in the middle line, arms hanging by the side in a relaxed condition (Fig. 49). Contraction of any of the muscles of the chest on one side may greatly interfere with the results. When the back is percussed the patient is asked to fold his arms each on the opposite shoulder with a view to removing the scapulæ as far outward as possible. With these bones in the normal position the

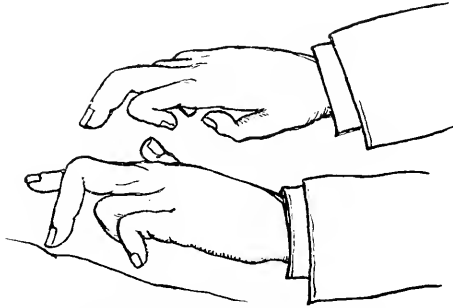


FIG. 48.—Hooked-finger percussion.

greater part of the lung in the supraspinous fossæ is beyond the bony thorax and the apex is partly covered by the shoulder-blades. To hammer away in the supraspinous fossæ, as we often see done, is a waste of time and energy, because percussion there strikes bone and thick muscles, and the waves, hardly, if at all, penetrate into the lung. But with folded arms, each over the opposite shoulder, or the patient embracing the back of a chair, the shoulder-blades are moved far away from the median line of the body, thus exposing the lung covered by comparatively thin parieties.

When it is desired to bring out the finer shades of resonance or, in doubtful cases, it is advisable to have the patient lying down on an upholstered couch or an examining table. Placing the patient with his back near a wall or door, or, as Lawrason Brown suggested, standing in the angle between two walls, may help in bringing out points which might otherwise escape attention.

<sup>1</sup> Münch. med. Wehnschr., 1902, xlix, 620.

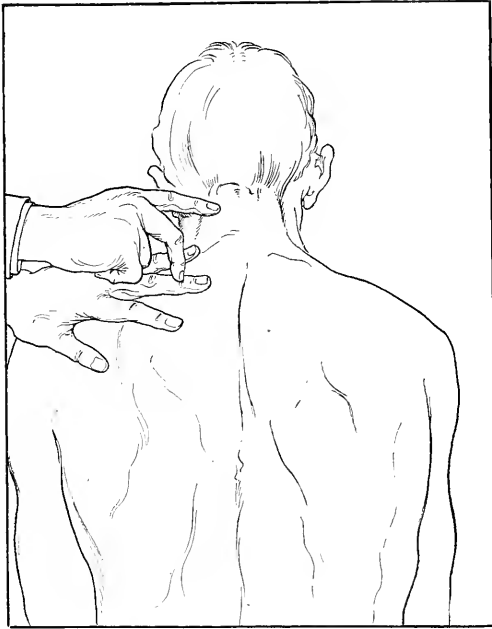


FIG. 49.—Percussion of the left apex posteriorly.

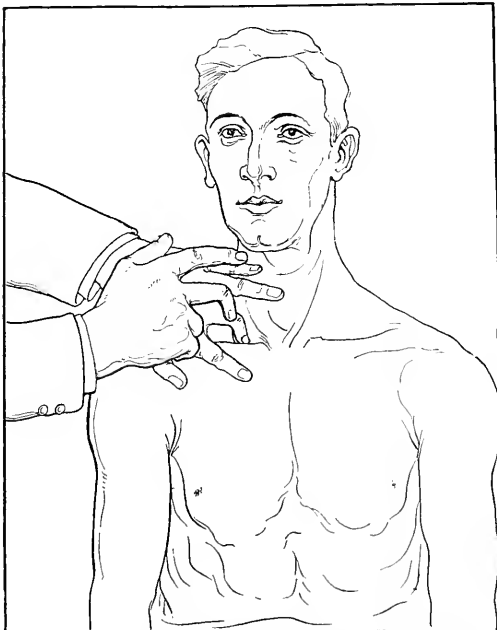


FIG. 50.—Hooked-finger percussion of the apex.

**Comparative Percussion.**—When percussing, we compare symmetrically corresponding areas on both sides of the chest and percuss with equal force while striking each side. This is especially important because there is no standard resonance for a healthy chest; every individual has his own resonance which depends on many factors, mainly the vibration of the chest walls and the contents of the thoracic cavity, which are inconstant values. But in the normal chest the resonance, as well as its qualities such as duration and pitch, are the same on both sides. The slightly impaired resonance over the upper part of the right side may be disregarded for practical purposes.

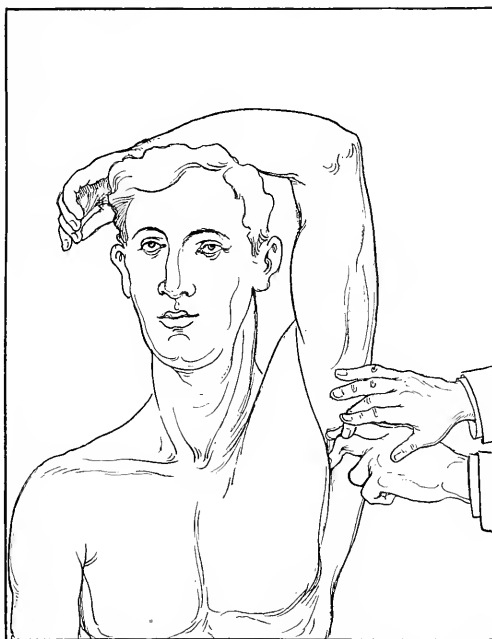


FIG. 51.—Percussion of the axilla.

In incipient cases there are “seats of election”—points where dulness is most likely to be encountered if there is an apical lesion. Anteriorly, it is mostly under the inner third of the clavicle and posteriorly at the inner margin of the upper half of the scapula.

A small area of defective resonance can often be discovered by immediate percussion directly over the clavicle, comparing one side with the other. Immediately above and below the clavicle mediate percussion will bring it out, if it is present. If, on light percussion, impairment of resonance is discovered, the force of the blow is diminished to a minimum, thus delimiting the affected area, and we can again percuss the same spot, gradually increasing the force of the blow, always having in mind the thickness of the integuments, with a view

to ascertaining the degree of dulness. If the dulness disappears with a heavy stroke, the lesion is of slight extent and superficial, or there may be a thickened pleura; but if it persists, we may feel confident that we are dealing with an extensive area of airless tissue.

Posteriorly, we look for dulness over the apices of the upper and lower lobes of the lung. The former is located in the supraspinous fossa near the spine and reaches the first thoracic spine; the latter is lower in the right side, reaches the fourth thoracic spine and higher in the left side at the third thoracic spine (Fig. 53). If impairment of resonance is present in incipient cases, it will be found at one of these four points.

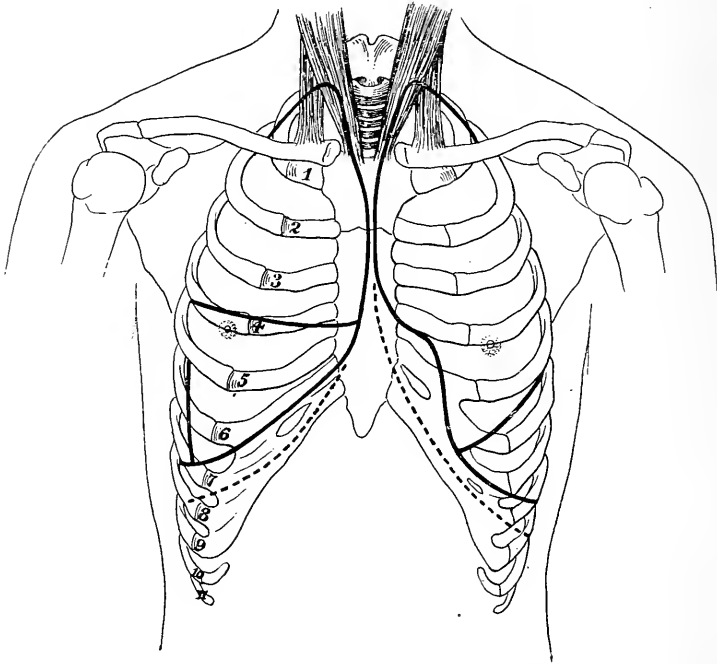


FIG. 52.—Lung margins according to Goldscheider.

While doing comparative percussion of apices it is imperative to remember that in the majority of healthy persons the resonance over the right apex above the third rib is somewhat defective, the note is shorter and of higher pitch. This has been attributed to various causes. The recent investigations of George Fetterolf and George W. Norris<sup>1</sup> have shown that it is due to the anterior position of the large vessels in relation to the right apex, as compared with the left; to the consequent encroachment upon, and reduction in size of, the right apex and to the contact of the inner surface of the right apex with the resonating trachea, while the left is in contact with non-resonating

<sup>1</sup> Amer. Jour. Med. Sci., 1912, cxliii, 637.

solid tissue. In right-sided lesions, when the signs are inconclusive, topographical percussion is therefore best.

**Tympanitic Resonance in Incipient Lesions.**—In the early stages the absence of distinct dullness in any part of the thorax is not always an indication of the absence of tuberculous infiltration. Impairment of resonance can only be brought out when the focus is at least one inch in diameter, although some, like Flint and Oestreich, have detected smaller foci. But small disseminated tubercles, before they become confluent, may alter the resonance in an altogether different direction. Causing relaxation or hyperfunction of the surrounding

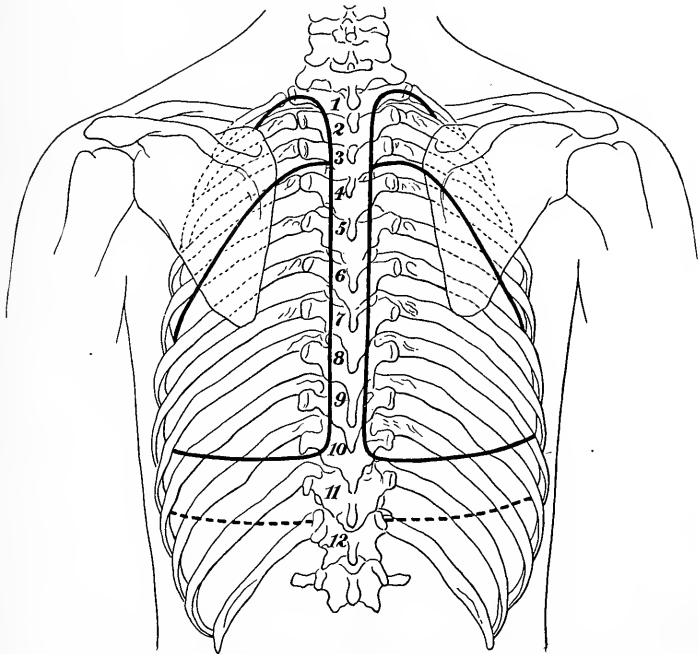


FIG. 53.—Lung margins according to Goldscheider.

lung tissue, they impart a tympanitic note on percussion. This tympany is of great importance in the diagnosis of incipient lesions, and is usually the cause why two competent observers will at times detect the lesion on different sides of the chest.

Everyone who has had the opportunities and inclination to watch incipient tuberculous lesions has met with cases in which the first sign obtained on percussion is localized tympany which subsequently changes into dullness with a tympanitic overnote, and finally becomes dull. Tympany in one suprascapular fossa, when accompanied by suspicious symptoms, is to be taken seriously; it may be the sole indication of small disseminated tubercles.

Absence of percussion signs, on the other hand, does not exclude incipient phthisis, because the lesion may be located deeply, subapically, or centrally, or it may be altogether a more malignant process—miliary or disseminated tubercles all over the lungs which have not yet become confluent. In the same manner, extensive tympany over one lobe, or one lung, with fever, cough, etc., may be an indication of extensive tubercularization of the affected part. The outlook is not as good as when the tubercles are localized in a limited area.

**Respiratory Percussion.**—In doubtful cases it is advisable to study the changes in the resonance during extreme and held inspiration and expiration, as was suggested by J. M. Da Costa<sup>1</sup> forty years ago. He showed that “at the apices, and especially in the infraclavicular region, in the suprascapular fossæ, and on a line toward the spine, a full held inspiration increases the resonance, makes the sound fuller and raises the pitch; and where, as is so common, the left side has normally a higher pitch, this disparity is preserved.” A held and complete expiration will greatly lessen the resonance and lower the pitch at the apices. “In the held inspiration we obtain a greater mass of tone; in held expiration, the reverse.” This change of resonance was found by Da Costa to remain unaffected in bronchitis; but in phthisis, even in the earlier stages, the affected area shows the reverse—a long, held inspiration gives a duller note than that observed on the healthy side.

This change of note during held inspiration and expiration is brought out very clearly by light percussion and is of great value in doubtful cases. When the infiltration increases in extent, involving the larger part of the apical parenchyma, the dullness on percussion is no longer modified by the forced and held expiration and inspiration. Hence we have in this method a very good test as to the extent of involvement in the tuberculous process. Aufrecht<sup>2</sup> confirmed these findings.

**Topographical Percussion of the Pulmonary Apices.**—There are cases of incipient phthisis in which comparative percussion gives no conclusive information, and only topographical percussion—mapping out the limits of the apical resonance—may clear up the case. This can only be done intelligently when we have clear ideas as to the limits of these resonant areas in the healthy person.

Krönig<sup>3</sup> showed that the resonant areas project as cones anteriorly and posteriorly, and that these two cones are united on the top of the shoulders by a narrow strip of resonance—the isthmus (Figs. 54 and 55). With careful and very light percussion we can easily map out the mesial line which runs in front, beginning at the sternoclavicular articulation, upward and outward forming a concavity inward, while posteriorly the line forms a convexity and ends at the level of the lower border of the second thoracic spinous process. The external line sep-

<sup>1</sup> Amer. Jour. Med. Sci., 1875, lxx, 17.

<sup>2</sup> Berl. klin. Wehnschr., 1912, xlix, 101.

<sup>3</sup> Deutsche Klinik, 1907, xi, 581 and 634.



arating the resonant apex from the dull shoulder and neck runs from the middle of the anterior border of the trapezius, curving downward and reaching the clavicle at the junction of the middle and outer third and continuing obliquely downward toward the axilla; proceeding upward, it forms a convexity toward the neck, crossing the shoulders, on the top of which it is separated from the mesial line by a resonant

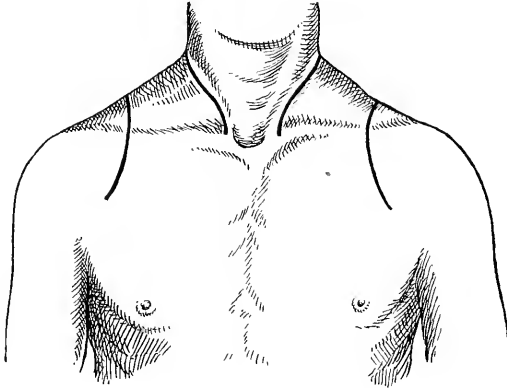


FIG. 54

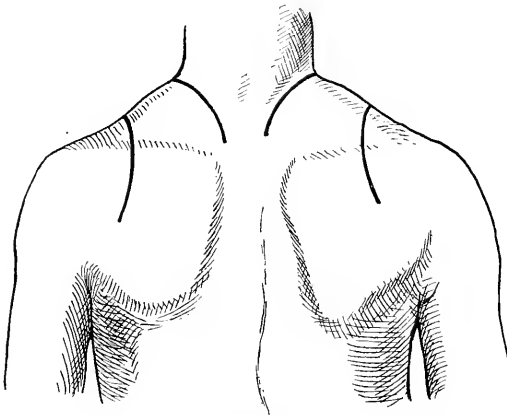


FIG. 55

Figs. 54 and 55.—Krönig's apical resonant areas.

space of about 2 to 3 cm. forming the isthmus, and proceeding downward with its concavity outward, terminating a couple of centimeters outside of the middle line of the scapula. Normally the height of the apex is anteriorly about 2 to 3 cm. above the clavicle, and posteriorly, on a level with the first thoracic spine, about 2 cm. outside of the middle line of the body.

It is important to remember that the pleximeter finger should be

applied parallel with the line we expect to delineate; in this case at right angles with the clavicle. It is also better to percuss from the lower parts of the chest upward, because in the former the normal note is usually found in early cases and it is always best to compare normal resonance with defective by striking the former first, as was already indicated.

**Changes in Apical Resonance in Phthisis.**—When the resonant areas are marked out on the chest of a healthy person, their height and width are practically the same on both sides. But in phthisis one side will be found contracted. Recalling that a tuberculous lesion in

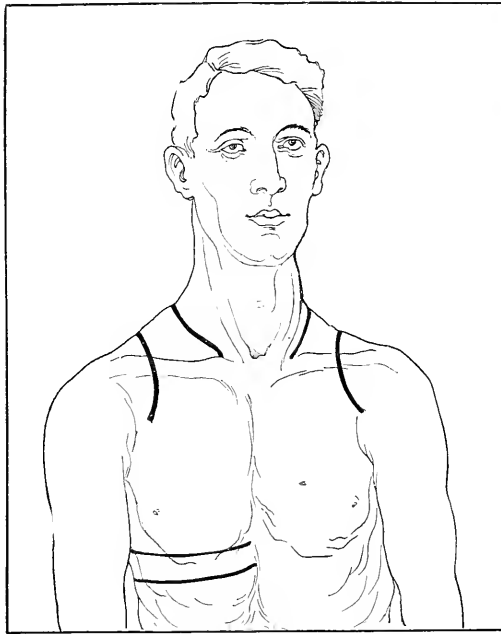


FIG. 56.—Contraction of the resonant area of the left apex.

the apex involves shrinkage of the pulmonary parenchyma, we have an explanation for this phenomenon. The extent of the shrinkage depends on many factors, mainly the degree of pulmonary retraction and the location of the lesion. When the lesion is centrally located, shrinkage of the apex is greater than when it is located at the periphery or under the pleura, as has been shown by Oestreich, obviously because in the former case traction is exerted on all sides. Autopsy findings show conclusively that this shrinkage occurs quite early, much earlier than is generally appreciated and for this reason we may get a clear view as to the condition of the lung in that region, by percussing the apices and mapping out Krönig's resonant areas.

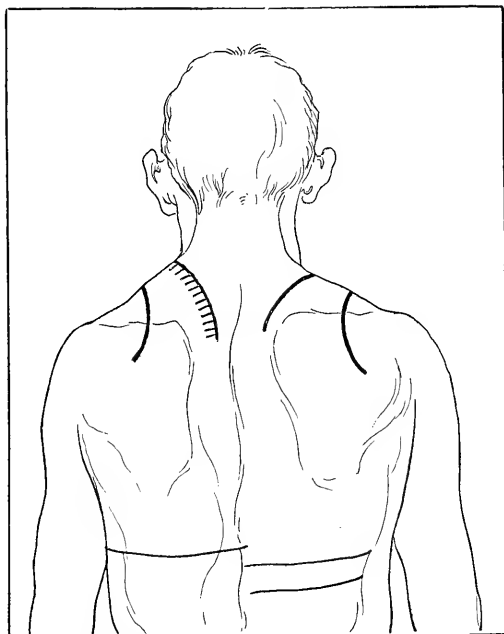


FIG. 57.—Krönig's resonant areas, showing a band of doubtful, or relative resonance at the mesial border of the left apex; also retraction of the lower margin of the left lung.

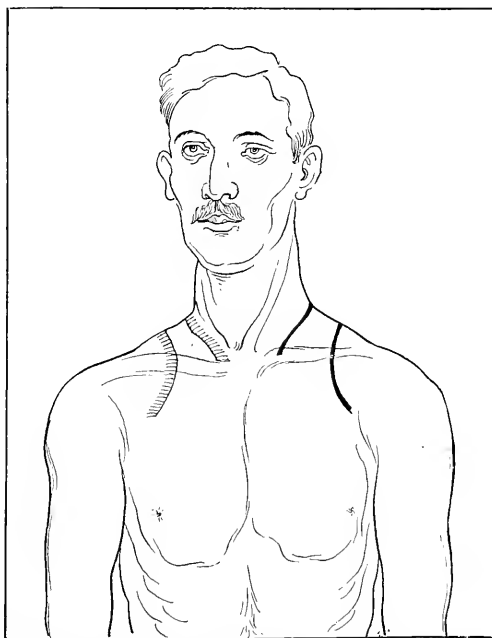


FIG. 58.—Bands of doubtful resonance on both sides of the right apex anteriorly.

Shrinkage manifests itself in two ways:

1. By a narrowing of the field of resonance on the affected side. This can be established by actual measurement. The isthmus in healthy persons is about two inches in width, and when we find it less than one inch in width, it requires investigation. The width of the base of the resonant cone may be measured simply in finger-breadths, as has been recommended by R. N. Philip.<sup>1</sup> Both sides are to be of the same width.

2. By a blurring of the line separating the resonant from the dull parts (Figs. 57 and 58). While in health we can easily percuss out a

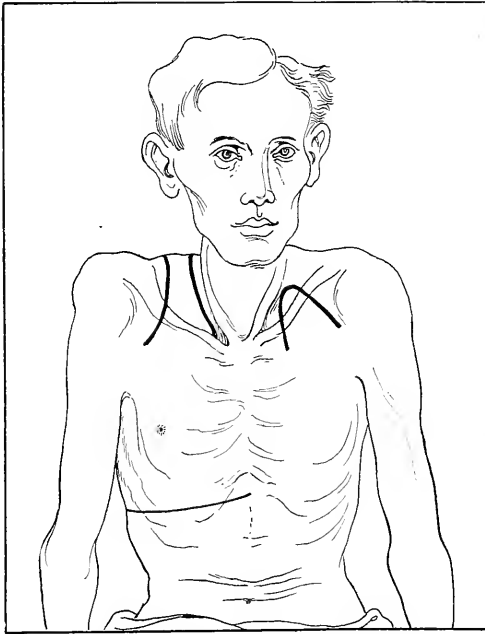


FIG. 59.—Frequent findings with Krönig's method of percussion in advanced cases. Retraction of the left lung.

clear line of demarcation, in tuberculous apices there is often an interval in which the resonance is doubtful. This is mostly found at the inner outline, but may be found at both sides. Krönig attributed it to changes in the tension of apical parenchyma at the margin of the affected parts. These points are better illustrated than described (Fig. 59), and in practice after the outlines of the apices have been marked out with a skin pencil, any existing differences in the outlines of the apices when one side is compared with the other are noted at a glance and need no measuring.

<sup>1</sup> Edinburgh Med. Jour., 1907, xxii, 473.

**Sources of Error.**—Krönig's method is of excellent service in most cases of incipient phthisis. But we often meet with cases in which after careful and time-consuming work, the results attained are unsatisfactory. I have seen cases of phthisis in which no dislocation of any of the outlines of the apical resonance could be made out. Then, there are numerous cases in which contraction of the apex is made out very nicely, but there is no active phthisis. This is especially true of "collapse induration," which will be discussed later on. Healed tuberculous lesions also leave contracted apices and what we seek to determine is the presence of active phthisis.

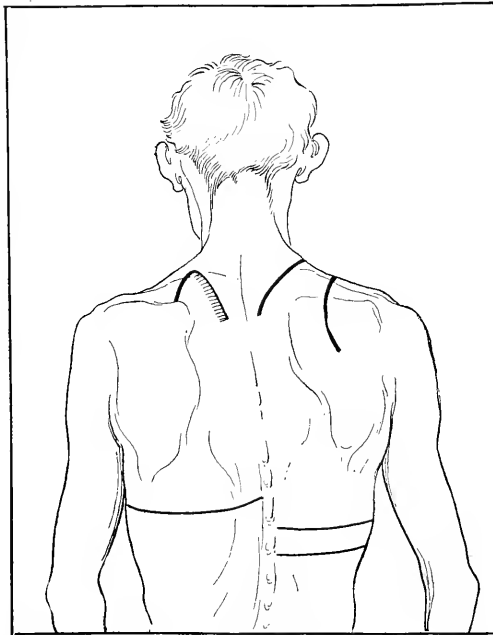


FIG. 60.—Same patient as in Fig. 59; findings posteriorly.

Krönig stated that in phthisis the motion of the base is invariably affected at an early stage, while in non-tuberculous apical lesions, the expansion of the lower margins of the lung remains normal. This does not hold in practice. There are many cases of phthisis in which the base retains its normal mobility during inspiration and expiration, and the reverse. The reason for the occasional failure of this method of percussion lies in the fact that the resonant area is not an outline of the true anatomical apex, but merely a projection of the same lung tissue in various directions (Figs. 61 and 62). The fact is that it is impossible to project the top of the lung on the surface of the body, considering its peculiar anatomical position and form. Krönig's isthmus, for instance, does not exist at all, and we must remember

that only the mesial border corresponds to the anatomical margin of the lung anteriorly and posteriorly. The lateral border cannot be determined with exactness in most cases because the percussion wave strikes the spot tangentially. In patients with marked scoliosis, the method is of no value at all.

**Goldscheider's Method of Apical Percussion.**—Anatomical studies by Goldscheider,<sup>1</sup> as well as orthodiagraphic examination of the lungs in their relation to the bony thorax, show conclusively that there is

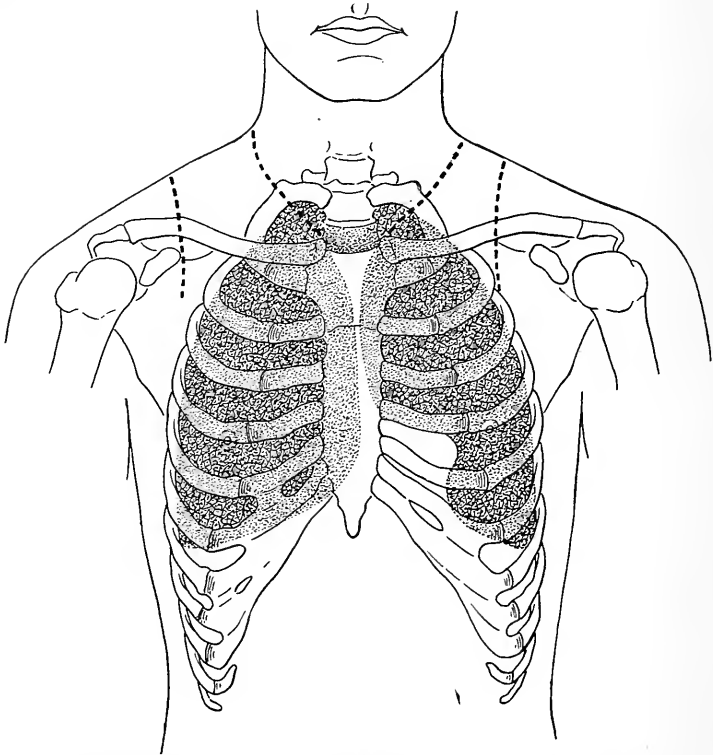


FIG. 61.—Showing that Krönig's resonant areas are not outlines of the apical margins, but are merely projections of the same lung tissue in various directions. (After Goldscheider.)

no lung tissue in most of the resonant area percussed out by Krönig's method. Anteriorly, the apex lies beneath the two heads of the sterno-cleidomastoid, protruding above the inner third of the clavicle for about one inch in height. This is seen clinically when emaciated persons cough and the lung is blown up above the clavicle, or in wasted infants during crying spells. Posteriorly, the apex of the lung lies close to the spinal column, reaching as high as the spinous process

<sup>1</sup> Berl. klin. Wchnschr., 1907, xl, 1267 and 1309.

of the first thoracic vertebra. But there it is impossible to obtain resonance from it because it is covered by a bony transverse process, rib and thick muscles.

Goldscheider,<sup>1</sup> for these anatomical reasons, devised another method of obtaining the resonance of the true anatomical apex, which we discussed in detail elsewhere.<sup>2</sup> From the complicated procedure of Goldscheider all that is of utility in doubtful cases is the determination of the height of the apex between the heads of the sternocleido-

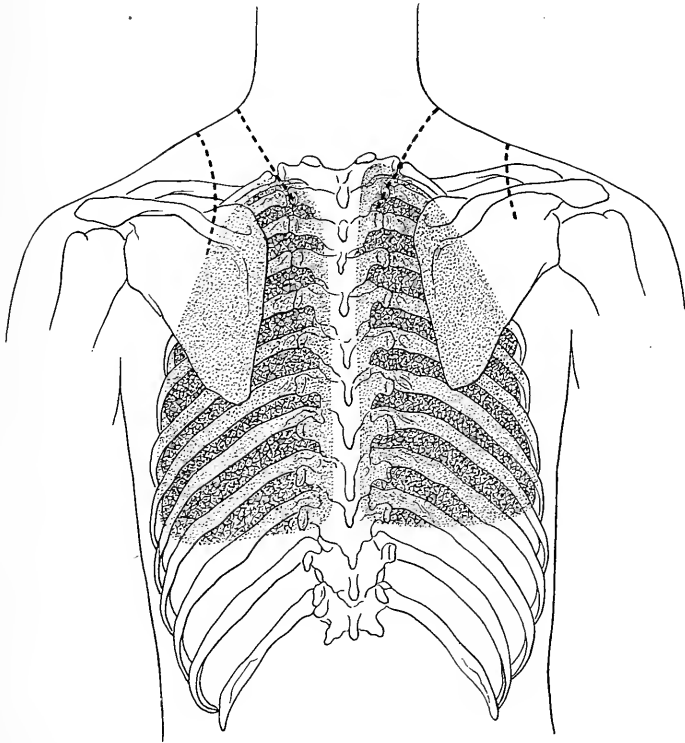


FIG. 62.—Showing that Krönig's resonant areas are not outlines of the apical margins, but are merely projections of the same lung tissue in various directions. In the supra-spinal fossæ there is no lung tissue at all. (After Goldscheider.)

mastoid, which can easily be done by percussing from below upward with the hooked finger as a pleximeter and comparing the two sides. Posteriorly, the lung resonance should reach the tip of the spinous process of the first thoracic vertebra on both sides. The height of the apices on both sides normally should be the same and if it is found shorter on one side it demands investigation as to the cause. In connection with other symptoms, it is strongly in favor of tuberculosis.

<sup>1</sup> *Ztschr. f. klin. Medizin.*, 1910, lxi, 205.

<sup>2</sup> *New York Med. Jour.*, 1913, xevii, 799.

**Pottenger's Views on Apical Percussion.**—There remains yet to mention that Pottenger sees utility in apical percussion mainly because we judge not only the resonance, but more so the resistance to the pleximeter finger. When there is an active and recent lesion in an apex the muscles above it are in spasm and offer greater resistance than normal muscles. He points out that when there is defective resonance because of an old and inactive lesion, the muscles are usually atrophied and feel soft and flabby to the pleximeter finger. Anteriorly, the sternocleidomastoid and scaleni and, posteriorly, the

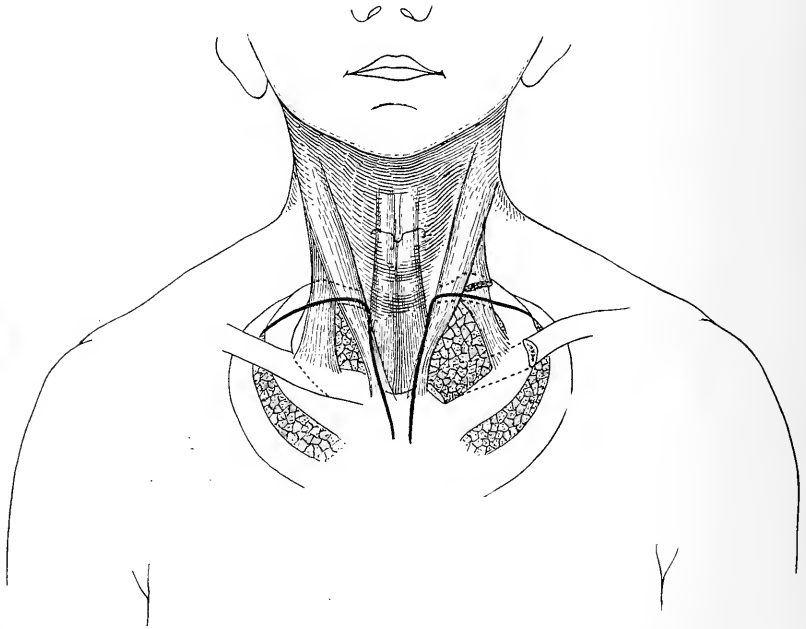


FIG. 63.—Topography of the apex according to Goldscheider: — upper and mesial borders of the lung; - - - - borders of the first rib and clavicle. On the left side the clavicular head of the sternocleidomastoid has been removed so that the scalenus anticus is visible. The upper border of the lung is somewhat higher than the first rib.

trapezius and levator anguli scapulæ are to be considered in this connection. This point has been of great assistance to me. Recently Galecki<sup>1</sup> has verified it in a very thorough study of the subject.

**Tidal Percussion.**—After ascertaining the limits of the apices, the base is to be delineated with a view to determining the vertical movements of the lung in the pleural sinus during both phases of respiration. This gives us information as to the presence or absence of emphysema, especially in fibroid phthisis, pleural adhesions, which are of such immense interest when thinking of applying a therapeutic pneumothorax, etc.

<sup>1</sup> Beitr. z. Klinik d. Tuberkulose, 1914, xxx, 363.



The lower margins of the lung resonance are first ascertained by percussion while the patient breathes normally and quietly, and marked with a dermatographic pencil. Then the patient is directed to take a deep breath and hold it as long as possible while we again percuss and ascertain the lower limits of the lung, and again mark them with the pencil. In healthy persons the difference in these two lines is between one and two and a half inches. It is to be borne in mind that on the left side the lung margin is naturally about an inch lower than on the right; also that the expansion is greater in the axillary line anteriorly than posteriorly. In emphysematous subjects, also in the senile, and in those with deformed chests, expansion may be very little or *nil*. Pain while breathing may have the same effect. On the left side, when there is no expansion anteriorly at Traube's semi-lunar space, it is an indication of pleural adhesions, or effusion; an increase in the tympany at that space indicates retraction of the left lung, not infrequent in phthisis.

In most cases of incipient phthisis the respiratory excursion of the affected lung is more or less restricted, and when there are adhesions, there is unilateral absence of respiratory excursions. But since we have been interested in pleural adhesions while making artificial pneumothorax, we find that these signs are not absolutely reliable.

**Percussion in Advanced Phthisis.**—With the advance of the disease the percussion findings become more and more varied and scattered all over the chest, and the difficulties of determining the exact condition of the lungs from percussion findings alone, more and more unsurmountable. The dulness elicited is usually due not only to the active lesions, but also to such as have healed or are quiescent; to thickened pleura, which is usually a conservative process; to pleural effusions, displacements of the heart, diaphragm, liver, stomach, etc. Some of these processes are permanent, others appear for a short time and disappear again. Localized emphysema, transient or permanent, due to vicarious function, often obscures deeply lying airless tissue.

In most cases, however, we find that one lung shows dense dulness in its upper part, usually as far as the third or fourth rib, as well as retraction of one or, more rarely, both bases. But even this may be due to healed or quiescent old lesions. We also find a frequent area of dulness in one and, at times, in both interscapular spaces due to lesions of the apices of the lower lobes, or enlarged glands. At times, the dulness runs along the lines of the interlobar fissures anteriorly and posteriorly. To map out such areas of dulness may be of scientific interest, but otherwise the diagnosis of these cases rests on other methods of exploration, especially the subjective symptoms. Signs of excavation are discussed elsewhere. (See Chapter XX.)

**Sources of Error in Signs Elicited by Percussion.**—When finding defective resonance over one apex, contraction of Krönig's resonant area on one side, or one apex shorter than the other, thus indicating pulmonary retraction, are we justified in considering the patient sick with active phthisis? Are differences in resonance elicited when the

two sides of the chest are symmetrically and comparatively percussed, especially in its upper third, sure indications of active phthisis?

These are problems that confront the clinician quite often and they can only be answered by an intelligent consideration of the causes of defective resonance and dullness, which are mainly airless lung tissue, and which may be due to many other causes in addition to tuberculosis. Besides, we may have differences in the resonance due to faulty technic in percussion, also because of asymmetry of the chest in cases of kyphosis or scoliosis, or unilateral hypertrophy of the muscles due to occupational effects. These factors are to be eliminated before we attempt to interpret percussion findings in early phthisis.

There are other sources of error. Chronic pneumonic processes, healed apical lesions and pleurisy are very common, as we have already shown, and many leave some airless tissue which is detected by careful percussion. So that even if due to tuberculosis, apical dullness or retraction does not always mean active phthisis requiring therapeutic intervention. Collapse induration, due to inhalation of dust in mouth-breathers, may show percussion signs which are undistinguishable from phthisis, if we should rely on percussion alone. We also occasionally find dullness in the apices in persons leading a sedentary life and who do not breathe deeply, especially chlorotic girls. Some of these cases are cleared up by directing the patient to breathe deeply for some minutes, or practising Da Costa's respiratory percussion.

We also meet now and then with persons in whom the resonance on one or both sides of the chest is defective without any excessive adiposity or strongly developed muscles to account for it. The air content of the lungs is less in childhood than in later life, and it decreases with old age, often without showing any anatomical changes in the lungs at the autopsy.

In many cases a study of the overlying muscles as to rigidity and atrophy has helped me immensely, while in others it was of no avail.

**Diagnostic Value of Percussion.**—In cases presenting symptoms of phthisis such as fever, cough, night sweats, etc., percussion findings alone are often sufficient to localize the lesion, and in many cases it will be found by prolonged observation that a lesion develops in the apex where we originally found only defective resonance or contraction of the field of resonance, though auscultatory signs were wanting.

Percussion findings alone, without any general symptoms of phthisis, prove nothing, just as in radiography a shadow over an apex does not prove an active tuberculous lesion. It is only in connection with the general symptoms that percussion, like any other single sign or symptom, can be utilized for diagnosis.

However, whenever found, defective resonance in an apex demands careful investigation and watching of the case, unless a reason is found for its existence.

## CHAPTER XVI.

### AUSCULTATION OF THE CHEST IN PHTHISIS.

WE have shown that percussion is a most valuable diagnostic method in early phthisis, even more valuable than in the later stages, and will often give definite information as to the air content of the lungs much earlier than other methods. Auscultation is just as valuable for other reasons. At times it affords information in cases in which the lesion is centrally located and in tuberculosis grafted on an emphysematous lung, when percussion and even skiagraphy may fail. Similarly, in advanced cases where the lesion is extending, altered breath sounds and rales may often be found in advance of dulness. On the other hand, acute cases, especially miliary tuberculosis, may show normal breath sounds and no rales, and in chronic cases with deeply lying cavities the normal lung tissue conceals all the signs of excavation. In the former diffuse tympany, while in the latter percussion or radiography, may disclose the exact state of affairs.

Believing that the technic of auscultation is much easier to master than that of percussion, many have discarded the latter and rely solely on the former, which is a grave error. The fact is that it is just as difficult to acquire skill in proper auscultation of the chest, and in interpreting the findings correctly, as to percuss properly. Some, like Goldscheider<sup>1</sup> and Clive Riviere,<sup>2</sup> believe that auscultation is even more difficult to master. It is because of faulty technic that auscultation does not yield all the information that can be obtained by this method.

**Technic of Auscultation.**—The patient should be stripped to the waist, just as for percussion, and seated on a high revolving stool, so as to be accessible from all sides. Before beginning auscultation the physician must assure himself that the patient knows how to breathe properly and if not, which is very often the case, proper instruction is to be given objectively. One important drawback to auscultation is that many patients do not know how to “expire”—they just inspire jerkily and stop with inflated chests. Others, usually such as have led a sedentary life and never expanded their chests properly, inspire and expire quickly and in rapid succession so that it is difficult to follow each phase of respiration. While in the vast majority a little instruction suffices, at times we meet with some, and not exclusively among those reputed to be ignorant, who will not breathe properly for our purposes, especially nervous individuals, and the examination must be postponed till they become accustomed to the physician.

<sup>1</sup> *Ztschr. f. klin. Medizin.*, 1910, lxi, 205.

<sup>2</sup> *Early Diagnosis of Tubercle*, London, 1914, p. 22.

The breathing must be regular, rhythmic, somewhat deeper than usual, and through the nose, because when the air enters this way the lungs expand much better and more uniformly. Mouth-breathing occasionally induces cough. In cases of nasal obstruction the patient breathes through his mouth, but we must guard against noises arising in the pharynx, especially those created by the soft palate, which impart a bronchial or blowing character to the breath sounds and, at times, give an impression of prolonged expiratory murmur, when in fact there is nothing of the kind.

Special attention should be paid to expiration, during which the patient should empty his chest as much as possible, without any undue exertion, and that each expiration should promptly be followed by a deep inspiration.

Any stethoscope to which the physician is accustomed may be used. The writer prefers the Bowles model. The bell should be applied carefully in the intercostal spaces, especially in emaciated persons, so that it makes an air-tight connection with the skin. It should be held firmly but without any undue pressure, thus excluding all extraneous noises. Movement of the bell of the stethoscope upon the surface of the body interferes greatly with proper auscultation and should be avoided.

**Single Phase Auscultation.**—To appreciate slight changes in the duration and quality of the respiratory murmur it is important to listen to each phase of the respiratory act separately. Grancher's<sup>1</sup> method has served me best. It consists in first listening to the inspiratory murmur and to neglect at the time the expiratory murmur; and when listening to the latter the former is to be neglected. Rales are always looked for separately, after we have a clear idea as to the character of the breath sounds.

Beginning, for instance, with auscultation of the left apex, we listen attentively to the inspiratory murmur, and while the patient expires, the bell of the stethoscope is quickly carried over to a corresponding point on the right side of the chest and we listen to an inspiration. The inspiratory murmur is thus compared right and left and any differences that may be found are carefully noted. In this manner the slightest change in the murmur on one side can be best appreciated, because we have a standard in the unaffected side. Only when both sides of the chest are affected is this method uninformative, because we do not have an immediate impression of a normal inspiratory murmur. The expiratory murmur is to be studied in the same manner, carrying over the bell of the stethoscope while the patient inspires, and noting the difference. While listening to these murmurs, no attention at all is paid to any adventitious sounds which may be present. These are left for separate study.

This method of auscultation, devised by Grancher, and hardly ever

<sup>1</sup> *Maladies de l'appareil respiratoire*, Paris, 1890.

mentioned in our text-books, is the only one that can bring out all the changes in the respiratory murmurs heard in really incipient pulmonary lesions, and should be used exclusively.

**The Normal Respiratory Murmurs.**—The most important prerequisite of proper interpretation of auscultatory findings in pathological conditions of the lungs is a knowledge of, and experience with, the respiratory murmurs audible in normal chests. Without this knowledge we cannot expect to appreciate slight changes audible during either phase of the respiratory act in early phthisis. It is because of the disregard of the qualities of the physiological breath sounds that slight changes are overlooked, and many state that only with the appearance of adventitious sounds can a positive diagnosis be made, which is decidedly wrong, just as is waiting for tubercle bacilli to make their appearance in the sputum. One who wants to appreciate the early changes of phthisis cannot auscultate normal chests too often.

The physiological or vesicular respiratory murmur shows that the pulmonary parenchyma at the auscultated area contains air which enters with each act of inspiration, and leaves with each act of expiration without meeting any obstruction in its course. During inspiration it is audible with different degrees of intensity all over the chest as a sighing, whispering rustle; during expiration there is either no murmur at all, or, more commonly, a very faint noise is heard which is somewhat lower pitched than the inspiratory murmur, notwithstanding that ~~inspiration~~ actually lasts longer than ~~expiration~~.

Without entering into the problem of the origin of these murmurs, whether they are produced in the glottis or in the air cells in the areas under examination, we want to emphasize that it is important to bear in mind while auscultating that any changes in pitch, quality and rhythm noted during either phase of respiration are to be given careful attention in cases in which early phthisis is suspected.

**Feeble Breathing.**—When meeting a patient with a really incipient lesion, which is not often our privilege because when they present themselves the lesion is usually more advanced than is generally appreciated, we find no adventitious sounds, no changes in the type of breathing, no bronchovesicular or bronchial breathing, etc. The most common change in the breath sounds at this stage is feeble breathing, or, more rarely, complete absence of the respiratory murmur over a circumscribed area in one of the apices, mostly found posteriorly near or above the spine of the scapula, the *zone d'alarme* of Sergeant,<sup>1</sup> and anteriorly beneath the inner third of the clavicle. At times this feeble murmur is blowing or even bronchial in character and at the end of inspirations some dry crackling may be heard.

To be of diagnostic significance this feeble breathing must be localized over one apex, circumscribed, fixed and persistent for some time, and uninfluenced by respiratory efforts and cough. It is an

<sup>1</sup> Le Monde Médical, 1912, xxii, 1121; La Clinique, 1913, viii, 437.

indication of peribronchial tuberculous infiltration compressing some bronchioles, thus creating atelectasis of the alveoli they supply; or of localized pleurisy interfering with the respiratory activity of the alveoli in the affected area.

Localized feeble breath sounds are also found over healed tuberculous lesions, or adhesions of the apical pleura following abortive tuberculosis. But during the early stage of active phthisis feeble breathing accompanies constitutional symptoms, such as cough, fever, tachycardia, etc., and usually some signs are elicited by percussion of the same area. As Bezançon<sup>1</sup> has pointed out, in the absence of constitutional symptoms, feeble breathing at one apex is a sign of a healed tuberculous lesion.

In advanced phthisis, we very often meet with limited areas of feeble or absent breathing, but vigorous cough removes the plug which obstructs the entry of air into a bronchus and breath sounds are again audible. It is noteworthy and of diagnostic importance that atelectasis is frequently produced by plugging of a bronchus and the resulting resorption of the air from the alveoli may produce dulness over the area supplied by that bronchus, but no breath sounds, no adventitious sounds are heard. Occurring at the base, it is often difficult to distinguish it from thickened or adherent pleura which is also characterized by feeble or absent breathing, as is pleural exudate.

In acute pneumonic phthisis I have repeatedly met feeble breath sounds in addition to dulness elicited over the affected lobe of the lung; at times there was even absence of all breath murmurs, but some moist subcrepitant rales were audible over the same region. Similarly, we may meet during febrile exacerbations in advanced cases, with feeble breathing over newly affected areas, which later changes into bronchial breathing, etc.

**Rough or Granular Breathing.**—This is often found in incipient cases. Here again it is the inspiratory murmur that is especially affected: It is dry, rough, low-pitched and, as Minor<sup>2</sup> describes it, it is made up of a succession of very short sounds, as though small, soft granules of fine wet sago were being rolled over each other. It should not be confounded with puerile or harsh breathing: Granular breathing may be altogether diminished in intensity, or even very faint, while puerile breathing is always intense and emphatically pure. On the other hand, in granular breathing there is always a suspicion that adventitious sounds or noises are superadding the inspiratory murmur. According to Sahli, it is a sign of bronchial catarrh; there is either partial impermeability of the bronchi producing unequal respiratory excursions of the affected lung area, or else the accompanying noises are derived from the secretions causing partial stenosis or irregularity in the lumen. When these accompanying noises can be plainly isolated, we call them rales, but as they remain indistinct

<sup>1</sup> Rev. de la tuberculose, 1913, x, 1.

<sup>2</sup> Klebs's Tuberculosis, p. 249.

and blended, the vesicular breathing becomes impure, granular or rough. It is generally heard over the suprascapular fossæ, or above and beneath the clavicle.

Grancher insists that granular breathing is a sure sign of incipient phthisis, and Clive Riviere speaks of it as the earliest auscultatory sign, while Piéry<sup>1</sup> says that it is nothing of the kind, but that it is a good sign of a cured lesion due to cicatrization of a limited area of lung tissue, which is undoubtedly a fact. I have seen many patients who presented granular breathing at an apex for years without showing any of the constitutional symptoms of phthisis. On the other hand, I have full confidence in this sign when there are the usual general symptoms of phthisis, because I have repeatedly observed that in the very area first presenting feeble or granular breathing, there subsequently developed typical lesions of phthisis. Of course, one must always bear in mind that the absence of constitutional symptoms is an indication that the granular breathing is probably due to a cicatrix remaining after a tuberculous lesion has healed.

**Interrupted or Cog-wheel Breathing.**—The *respiration saccadée* of the French, is another anomalous type of breath sounds which has for a long time been considered characteristic of early phthisis. The inspiratory murmur is not smooth and continuous as in normal respiration, but is broken, so that it appears jerky, divided into several more or less distinct parts. It differs from rough breathing by the fact that each portion of the sound retains its smooth, rustling character. It is apparently caused by the obstacles met by the air current while entering the alveoli. The breath sounds may be increased or, more commonly, decreased in intensity.

I find cog-wheel respiration only rarely a sign of incipient phthisis and am inclined to agree with Piéry who says that in the region of the apex it is always an indication of pleural adhesions which are often the remains of a healed tuberculous lesion. In some cases, however, it is met with in the beginning of active phthisis and the fact that in the later stages of the disease it can very often be heard along the borders of advancing lesions, shows that the factors producing it may be of the first disturbances of the respiratory murmur in the areas of impaired breathing capacity around infiltrated portions of the lung.

Cog-wheel breathing is occasionally heard over chests in nervous patients or such as have pains due to acute pleurisy, or who shiver during the examination. But then it is heard all over the chest, while in phthisis it is localized over a limited area.

**Prolonged Expiration.**—From what has been stated it is evident that in the very early stages of phthisis, auscultation reveals only changes in the inspiratory murmur, a point which cannot be too

<sup>1</sup> La tuberculose pulmonaire, Paris, 1910, p. 311.

strongly emphasized. In older books on the subject we almost always read that changes in the expiratory murmur are pathognomonic of early phthisis, obviously because in former days incipient phthisis, as we know it today, was not recognized. In fact, because even today patients only rarely present themselves for examination at the very incipiency of the disease, we usually find a prolonged expiratory murmur at the first examination. But speaking as one who has had opportunities for examination of large numbers of persons who do not even suspect that they have any pulmonary trouble, and examining the lungs of everyone who comes under my care, I find that changes in the inspiratory murmur, such as feeble breath sounds, rough or cog-wheel breathing, are usually found earlier than changes in the expiratory murmur.

In normal vesicular breathing the expiratory murmur is either inaudible or, more commonly, it lasts only one-fifth to one-fourth the time of the inspiratory murmur. When it lasts as long as, or longer than, the inspiratory murmur it is undoubtedly pathological, though not necessarily of tuberculous origin. When audible all over the chest it is an indication of bronchitis or pulmonary emphysema, but when we find it localized at one apex, its significance as a sign of phthisis is to be appreciated. It may be due to sclerosis of a limited portion of the lung tissue, as is the case in healed tuberculous lesions. Indeed, when it also has a bronchial timber it is pathognomonic of this condition, and Turban speaks of it as "cicatrical respiration."

In active early lesions, prolonged expiratory murmur localized at an apex is an indication of either catarrh of the smaller bronchioles, or pressure on these tubes in cases in which infiltrations produce stenosis. It is therefore usually met with later than the changes in the inspiratory murmur, of which we spoke above. The prolonged expiratory murmur is often harsh and rough, and with the advance of the disease it gradually acquires a bronchial character, finally becoming pure bronchial or tubular breathing. While we may meet it without any adventitious sounds, this is exceptional in my experience. On the other hand, it may be feeble and hardly audible and at times we hear the rales very clearly while the prolonged expiration is only detected after careful listening.

There is another fact to be borne in mind while evaluating prolonged expiration as a sign of early phthisis. Not only may it be the only indication of a healed lesion, as has already been stated, but in the right apex it may not be due to tuberculosis at all, especially in young adults with thin thoracic walls. In collapse induration it is not uncommon, while in persons working at dusty trades, such as stone-cutters, carpenters, miners, garment-workers, etc., the expiratory murmur at the right apex is very often harsh, rough and prolonged. Under the circumstances it is of more significance when found in the left apex, and in the right side a careful study of the constitutional symptoms must be made before attaching any diagnostic value to it.



**Bronchial Breathing.**—With the advance of the disease the disseminated tubercles in the lung conglomerate by growth and form a solid circumscribed mass over which the breath sounds elicited on auscultation are more or less characteristic. The vesicular quality of the murmur changes by degrees till it finally becomes high-pitched, clear and blowing during both inspiration and expiration, which is very prolonged.

Bronchial breathing is a sign of consolidation of lung tissue: The laryngotracheal murmur is transmitted and, according to Sahli, even magnified while passing from the bronchi through consolidated lung tissue to the surface. It is thus heard over areas which are dull on percussion, particularly over the upper third of the chest anteriorly and posteriorly. During the course of chronic phthisis bronchial breathing is also caused by many complications which produce compression of the alveoli with resulting pulmonary atelectasis, as is the case in pleural effusions, pneumothorax, hydrothorax, etc. In these cases the bronchial breathing is engendered only when the alveoli and, at most, the bronchioles are compressed; when the large tubes are also obliterated by compression no breath sounds at all are audible.

In acute phthisis, bronchial breathing is mainly caused by caseous infiltration of the affected areas, and it is harsher, louder and more high-pitched, the more compact and extensive the consolidation of lung tissue. Bronchial breathing in phthisis is not as loud and resonating as in pneumonia, and when it is encountered it is an indication of an acute process which is probably progressive and of serious prognostic significance. It is therefore found early in the disease in acute pneumonic phthisis and during chronic phthisis over the seat of new extensions of the process involving the larger part of a lobe, and in the terminal stages when pneumonia complicates an old lesion and carries off the patient. In chronic phthisis, the higher the pitch of bronchial breathing, the greater the consolidation of lung tissue which may be assumed.

It is a fact to be remembered that in the average case of chronic phthisis bronchial breathing does not appear suddenly, but by slow degrees. The vesicular murmur is gradually transformed into broncho-vesicular, which, with the subsequent consolidation of the process, finally becomes purely bronchial.

**Bronchovesicular Breathing.**—On rare occasions, we may find bronchial breathing without dulness over the same area; in fact, I have at times met it over areas emitting tympanitic resonance on percussion, which is an indication that even small disseminated tubercles, which are incapable of producing dulness, but relax the lung tissue and cause tympany, may cause bronchial breathing.

But usually disseminated tubercles produce bronchovesicular breathing. We hear a mixture of both vesicular and bronchial sounds over the same area, the former originating in the small consolidated areas which transmit the laryngotracheal sounds, while the latter come from

the alveoli of the unaffected lung tissue that surrounds the tubercles. It is thus clear that the presence of bronchovesicular breathing is an indication of small tubercles scattered within normal lung tissue. This is usually preceded by prolonged expiration which changes by degrees into bronchovesicular breathing and finally into bronchial, as has already been shown.

**Sources of Error.**—Bronchial and bronchovesicular breathing *per se* are not indications of phthisis. In addition to the many pathological conditions which may cause this type of breath sounds, we quite often hear it over healthy chests. There are many individuals in whom bronchial breathing is heard all over the upper parts of the thorax. In the interscapular, right supraspinous and supraclavicular spaces it is very common in apparently healthy persons, especially during vigorous breathing. This is said by Bandelier and Röpke to be found in about one-third of healthy people; it is due to differences in the anatomical structure of the two apices. Fetterolf and Norris<sup>1</sup> have studied these differences in structure in detail and it appears that the breath sounds have better opportunities for transmission to the surface on the right side than on the left. In addition, because the right lung has three main bronchi, it favors the transmission of bronchial breathing more than the left, which has only two.

Bronchial breathing is very common in these locations and is not to be given undue diagnostic significance unless there are other symptoms and signs of phthisis. Individuals with thin thoracic walls are more apt to show this sort of breath sounds, while vigorous breathing and dyspnea may accentuate it. To be of diagnostic significance, bronchial breathing must be strictly localized over a limited area and accompanied by other physical signs, especially dulness at the same spot.

Another source of error in auscultation is the frequent changes we meet in the respiratory sounds in many patients. One day we meet at the affected area bronchial breathing and the next day we are surprised by vesicular, or feeble breathing, or complete absence of breath sounds over the very area where distinct pathological auscultatory phenomena were audible the day before. Vigorous cough, by removing the mucous plug in some tube, may reestablish the original sounds. I have seen such changes occurring during an examination which lasted less than half an hour. We should therefore beware of pronouncing a patient free from changes in the breath sounds before making him cough, and reexamining the chest on several different days.

Cavernous and amphoric breathing are discussed later when speaking of pulmonary excavations and of pneumothorax.

**Adventitious Sounds.**—As was already stated while speaking of the technic of auscultation, adventitious sounds are to be looked for only

<sup>1</sup> Amer. Jour. Med. Sci., 1912, cxliii, 637; Fetterolf, Arch. Intern. Med., 1909, iii, 13.

after ascertaining the character of the breath sounds during each phase of the respiratory act. To pass judgment at one time about both breath sounds and rales is hazardous and we are liable to overlook many important points which are of diagnostic and prognostic significance.

The adventitious sounds audible over phthisical chests in the various stages of the disease are manifold. It can be stated that all kinds of rales—sonorous, sibilant, crepitant, subcrepitant, gurgling, etc.—are met with during the course of the disease, and each variety has some significance, indicating the pathological condition of the lung. Paradoxical though it may seem at first sight, yet it is a fact that there is no rale which is pathognomonic of phthisis, nor does their absence exclude the disease. Especially is this true of the very incipency of active phthisis which, as was already intimated, begins as an infiltration and not as a catarrh of the bronchi. The neoplastic peribronchial formations may compress the alveoli; the proliferated interstitial tissues may contract and obliterate some air cells, etc., but such processes do not produce rales because at this stage the bronchi are not flooded with fluid or semifluid secretions which could interfere with the entry or exit of air through the bronchioles and air cells. Moreover, around an infiltrated area the lung usually acts vicariously and thus veils any alteration in the breath sounds that may be created in the diseased focus and the most we may expect is feeble, harsh or cog-wheel breathing, but no rales.

Rales are only produced when the caseous material softens and breaks through the walls of a bronchus: The secretions may irritate the bronchial mucous membrane and produce a catarrh which, in its turn, produces more secretion which, when set in motion by the passing air stream, engenders rales. This is a fact that I have had many opportunities to observe in patients who at first showed only alterations in the breath sounds, especially weak vesicular murmur or cog-wheel breathing, etc., but no rales, in spite of all constitutional symptoms of phthisis which went on its course, and only later adventitious sounds made their appearance. In such cases a diagnosis of phthisis must be made without finding any rales. In fact, I have met with acute cases in which a whole lobe was infiltrated in a comparatively short time; percussion showed distinct dulness, auscultation disclosed prolonged expiration, even bronchial breathing, but no rales at all were audible. It will therefore bear repetition that waiting for rales, as some text-books teach, may be worse than waiting for tubercle bacilli in the sputum before making a diagnosis.

**Creptitation.**—With the onset of softening, the crepitant and, at times, the subcrepitant rale can be discovered at the affected area. The former is audible exclusively during inspiration, or only at its end, and has been compared to the sound produced by rolling one's hair between the fingers near the ear. All agree that this rale is not caused by the motion of fluid secretions in the small bronchi and air

cells; nor by the explosion of air bubbles in the bronchi, as was formerly supposed. The consensus of opinion appears to be that it is caused by the inspiratory stream of air tearing apart sticky surfaces of the approximated alveolar walls, though many hold that the crepitant rale is altogether a friction sound produced by rubbing of the pleura covered with tubercles, as was first suggested by Leaming.<sup>1</sup> I am inclined to consider them purely atelectatic rales analogous to those met with over the margins of healthy lungs in persons who breathe superficially, and which are often mistaken for crepitations.

Crepitant rales are usually audible during quiet breathing, and provoked by vigorous coughing and breathing. Moreover, they disappear after several strong efforts at deep breathing, which would not be the case if they were friction sounds. They may be found early in the morning and missed throughout the day, and I have seen them appear and disappear within half an hour during an examination. At times, they are heard at a very early stage of the disease as quite numerous cracklings over the affected area, while in other cases but few are audible and they are spoken of as "dry crackles," the *craquements secs* of French authors.

Crepitant rales are not by any means pathognomonic of phthisis, for reasons already stated, but when audible over an apex showing contraction of Krönig's resonant areas, or impaired resonance in a person showing some of the important constitutional symptoms of phthisis, they are to be taken seriously. However, in order to evaluate them properly, we must carefully study them with particular reference as to permanence during several examinations on different days and that cough does not entirely remove them. I attach greater significance to crepitant rales when heard over the suprascapular fossa, the alarm zone (see p. 313) than when heard anteriorly above or immediately below the clavicle, because in the latter location they are as often spurious as real. We are often able to follow them up to the stage when they become moist—subcrepitant—and finally we find that signs of excavation appear at the same spot.

During the course of phthisis, the crepitant rale is heard quite often around the seat of the main lesion, indicating that the process is extending, and over pneumonic areas so often caused by acute exacerbations. In unilateral cases in which the other side is secondarily implicated, we may find that in the latter the first audible adventitious sounds are crepitations and these secondary lesions are worthy of study by those who want to be able to recognize and evaluate these adventitious sounds. In fact, while teaching tuberculosis to students, advanced cases are better for this reason than early cases in which the diagnosis is often doubtful.

**Moist Rales.**—With the advances of the process, softening sets in and the disintegrated tubercles are eliminated from the focus

<sup>1</sup> Diseases of the Heart and Lungs, New York, 1884.

through the bronchi, to be finally expectorated. These fluid and semifluid secretions, while remaining at the site of the lesion and in the bronchi, are often obstacles to the entry and exit of the air current and thus produce rales. In mild cases with but little secretion, we meet with the high-pitched subcrepitant rales produced in the small bronchi. When softening and liquefaction proceeds and the secretions become more and more copious, the size of the rales increases and we hear medium, large and coarse bubbling rales and gurgles.

The difference in the size of the rales apparently depends on the difference in the size of the bronchi in which they originate—large bronchi can hold larger masses of fluid and mucous secretion, and in smaller tubes less secretions are moved, while in excavations the mass of secretion may be very large and, as a result, we get gurgles. The larger rales are more intense and louder, though of a lower pitch than the smaller, but the latter are usually more numerous, evidently because there are more small bronchi than large ones. Rales are greater in number and more consonating when originating superficially, while those engendered deeply in the lung may not be heard at all. At times, we can hear rales in central lesions by placing the bell of the stethoscope in front of the patient's mouth, while all over the chest nothing is audible.

It must be emphasized that no rales *per se* are pathognomonic of phthisis, because we hear more adventitious sounds in many other conditions, notably bronchitis and bronchiectasis, than in the average case of chronic phthisis. To be of significance, the rales must be strictly localized over a limited area and persistent. It can be stated that, excepting in far-advanced cases, or the rare cases of chronic bronchitis complicating tuberculosis, and some forms of fibroid phthisis, the larger the area over which moist rales are heard, especially bilaterally, the less the likelihood of their being of tuberculous origin; the higher up in the chest they are exclusively audible, the more likely that they spell phthisis; and, when heard exclusively at the bases or over the lower lobes, the chances that they are tuberculous are rather scanty. Large bubbling rales, when heard over areas where there are no large bronchi, as in the upper third of the chest, are of greater significance than when heard over areas beneath which large bronchi are located. The latter may be caused by bronchitis or bronchiectasis. When large bubbling rales are heard near the bell of the stethoscope, they are indications of phthisical excavation, because there are no large bronchi near the surface of the lung.

**Sibilant and Sonorous Rales.**—These are very often heard over tuberculous foci. In many incipient cases, especially in those with stationary or healing lesions, whistling and snoring rales are not uncommonly localized over one apex, especially posteriorly. When not accompanied by crackles we may take them as an indication of healing and that they are caused by the compression of the bronchioles by fibrous tissue which forms during the process of repair. Similarly,

we hear sibilant and sonorous rales as the only reminders of an old and cured tuberculous process. In senile phthisis, sibilant and sonorous rales are often the only adventitious sounds.

The asthmatic forms of phthisis, as well as those accompanied by, or implanted on, diffuse bronchitis and pulmonary emphysema, especially in fibroid phthisis, often manifest themselves by sibilant and musical rales heard during inspiration and expiration. We hear all kinds of musical notes, snoring, cooing, whistling, grunting, groaning, whining, etc. They may be heard alone while the respiratory murmur is feeble or inaudible, and then they may also be accompanied by all kinds of moist rales. When audible all over both sides of the chest, the diagnosis of tuberculosis may not be an easy task and differentiation from chronic bronchitis, pulmonary emphysema, asthma, etc., can only be made after considering the signs revealed by percussion, as well as by the constitutional symptoms, and in some cases only the microscopic findings in the sputum can decide. When these sonorous and sibilant rales are heard unilaterally they are easily diagnosed as a rule.

**Friction Sounds.**—These are very often heard over phthical chests. Over the apex they are heard best anteriorly above and beneath the clavicle, but here they are usually not very distinct because of the limitation of the motion of the lung in that region. Yet, we sometimes perceive some grating. This is usually very difficult to differentiate from crepitation—all the criteria given in text-books are futile in some cases. At the lower parts of the thorax friction sounds are more common, especially in the axillary region. On rare occasions, a pleuropericardial rub is heard not only during the respiratory phases, but also synchronous with the heart beat. It is an indication of dry pleurisy of the lingula or other parts of the pleura in contact with the pericardium.

We distinguish friction sounds from rales by the fact that the former are heard superficially, right near the bell of the stethoscope; often they are increased by pressure of the stethoscope; they are uninfluenced by cough which usually increases the intensity of rales or entirely removes them; they are annulled when the breath is held. But the most important difference is that crepitant rales are heard during inspiration only, while friction sounds are audible during both phases of the respiratory act. However, in many cases it is quite difficult to state positively whether the adventitious sounds under consideration are of pulmonary or pleuritic origin. When found over an extensive area, especially posteriorly or in the axillary region, frictions may be diagnosed by assuming that rales over such a large area would represent a very extensive pulmonary lesion with severe constitutional symptoms, while pleurisy may persist for years without impairing the general condition of the patient very much.

**Spurious Rales.**—Rales of extrapulmonary origin are occasionally heard while auscultating chests, and attributed to tuberculous changes

in the lungs. In persons suffering nasal obstruction we may hear various sounds resembling rales which disappear when the patient is made to breathe through the mouth. A frequent cause of extrapulmonary rales is the falling back of the tongue when the patient makes strong efforts to breathe deeply, also after vigorous coughing the patient swallows and we believe that we hear rales in the chest.

Other spurious rales, described by Peretz<sup>1</sup> and William Ewart<sup>2</sup> in England, and Bushnell<sup>3</sup> and Hawes<sup>4</sup> in this country, are caused by muscular contractions, especially the trapezius, and on raising and lowering the shoulders and arms. In persons who lift their shoulders when asked to breathe deeply these "rales" are often quite audible. French authors speak of them as *craquements et frottements sousscapulaires*, which can be heard very often over the upper part of the chest posteriorly. These muscle sounds were a potential source of error in 9.2 per cent. of 250 cases examined by Hawes, while joint sounds were found in 22 per cent. of cases.

The so-called atelectatic and marginal rales are even more often found and must be guarded against. They are mostly heard over the anterior and lower margins of the lungs and are probably caused by the unfolding of collapsed alveoli in individuals who breathe superficially and also by the peeling off of the diaphragm from the chest wall as the lung descends into the complementary space. Richard C. Cabot<sup>5</sup> found them in 61 per cent. of normal chests and speaks of them as of crepitant and subcrepitant varieties. They usually disappear after a few breaths, but at times they persist indefinitely.

Bushnell also describes sounds originating in the sternum and its articulations, heard particularly at the second costal cartilage, which may lead to error, and I have been able to verify his findings in a large number of healthy persons, especially muscular men. In some cases they resemble crepitation and occasionally even medium-sized moist rales and clicks, like the adventitious sounds of early phthisis. They can usually be differentiated from pulmonary rales by the fact that they are localized and heard loudest over the sternum and its articulations, but in doubtful cases, especially those showing a short note at one apex, they may lead to error.

**Voice Sounds.**—Bronchophony adds little if anything to the information we gain by percussion and auscultation. It is generally heard over areas which are dull on percussion and show bronchial breathing. Moreover, it is necessary that the pulmonary consolidation should be superficial in order to produce distinct bronchophony while the breath sounds may be altered with moderately deep lesions. Of course, loud transmission of the voice suggests dense pulmonary consolidation through which a bronchus is passing, while decreased voice

<sup>1</sup> Brit. Med. Jour., 1896, i, 82.

<sup>2</sup> Ibid., 1912, i, 771.

<sup>3</sup> Medical Record, 1912, lxxxii, 101; lxxxii, 1109.

<sup>4</sup> Boston Med. and Surg. Jour., 1914, clxx, 153.

<sup>5</sup> Physical Diagnosis, New York, 1909, p. 163.

sounds indicate pleural effusions, thickened pleura, emphysema or even thick chest walls; in short, anything that diminishes the conductivity of the lung and intervenes between the large bronchi and the surface. Even a plugged bronchus may diminish or abolish the voice sounds, which reappear after several vigorous coughs.

Bronchophony is very loud in persons with thin chest walls, or who have a deep voice; and in general, in the interscapular space, especially in the right side, for obvious reasons. The various distinctions of bronchophony, pectoriloquy, etc., have no significance in the diagnosis of phthisis.

**Whispered Voice.**—Of greater importance is the auscultation of the whispered voice. In this it is really not the voice that is transmitted, but the breath sounds to which are added different reverberations from the oral, pharyngeal, and nasal cavities. My experience is in agreement with that of Sewall to the effect that in auscultation of the whispered voice we have an unrivalled means for the detection of minute changes in the pulmonary tissue. I have been able to outline consolidations and excavations of lung tissue by carefully studying the whispered voice, and other methods of diagnosis have merely confirmed the findings. Inasmuch as it is very easy to acquire, it ought to be more generally adapted in the routine study of phthisis in all its stages.

We must, however, remember that the chest walls are also vibrating when the person whispers and, especially, when he talks, as has been shown by Sewall.<sup>1</sup> He suggested that the mural vibrations should be damped by pressure with the stethoscope, and thus only the visceral vibrations will be brought to the auscultating ear. He shows that, in general, it may be said that with the intense congestion of the lungs or such tissue changes as occur in early phthisis, the voice takes on a more or less amphoric or tracheal character and it tends to become more distinct, prolonged, raised in pitch, and nearer the ear with pressure of the stethoscope on the surface of the chest. When the patient counts "one, two, three," there is a tendency for the voice to linger with a bleating echo which is exaggerated by stethoscope pressure. This has often helped me in doubtful cases in which both percussion and auscultation were absolutely inadequate to justify a final opinion.

Whispered pectoriloquy is also of immense value in patients with laryngeal involvement, or who have pleural pains and cannot breathe deeply, and especially in patients soon after a hemorrhage when we should hesitate in going through all the diagnostic maneuvers which may cause the bleeding to recur. Whispered pectoriloquy and bronchophony and auscultation during ordinary breathing can give us sufficient information to form an opinion on the extent of the lesion.

Over healthy lungs the whispered voice is audible in the upper third

<sup>1</sup> Jour. Amer. Med. Assn., 1913, ix, 2027; Sewall and Childs, Arch. Intern. Med., 1912, x, 45.



of the chest, especially on the right side, while in the lower parts it is hardly or not at all audible. An increase in the intensity is an indication of better sound conduction—consolidation or compression of pulmonary parenchyma, or even congestion, as has already been mentioned. It is therefore an early sign of phthisis. It must, however, be borne in mind that it is heard over healed lesions and therefore is not to be taken for a sign of activity of the process without confirmation by constitutional symptoms.

Over air-filled cavities, pulmonary or pleural, we hear what Kuthy<sup>1</sup> calls “amphorophony”—the transmission of the whispered voice with an amphoric or metallic echo. It is an indication that the cavity or pneumothorax has smooth walls. In cases with cavities we can at times make out the extent of the excavation by auscultation of the whispered voice as well as by any other method.

<sup>1</sup> Die Prognosenstellung bei der Lungentuberkulose, Berlin, 1914, p. 302.

## CHAPTER XVII.

### SKIAGRAPHY IN THE DIAGNOSIS OF PHTHISIS.

Soon after the introduction of the  $x$ -rays, great hopes were entertained that finally a means of visualizing the condition of the thoracic viscera and detecting any changes in the lungs, bronchi and pleura had been obtained. But after several years' experience it was found that in tuberculosis skiagraphy has its limitations, just as other diagnostic methods. On the one hand, it does not disclose infiltrations, the very early changes in phthisis; on the other hand, because it clearly shows caseated and calcified foci, revealing airless areas of lung tissue, it helps in establishing an anatomical diagnosis. Whether the changes discovered are tuberculous in character, and whether the lesion is active, must be ascertained by other clinical methods. For this reason, skiagraphy, while a very important aid in diagnosis, cannot be relied on to the exclusion of other methods. It does not disclose catarrhal conditions, nor does it reveal infiltrations.

When properly used skiagraphy helps materially in discovering certain changes in the intrathoracic viscera which formerly escaped notice during the life of the patient. Especially is this true of deep-seated lesions, pleural adhesions, enlarged bronchial glands, localized and interlobar effusions, localized pneumothorax, small cavities in the lungs, the motion of the diaphragm, abscess and gangrene of the lung, etc.

The condition of the lung and the changes at the site of the lesion in the average case of early phthisis can be made out easily by auscultation and percussion. The former even gives important indications as to the activity of the process discovered. But the  $x$ -rays complete the examination, and often reveal deeper-lying changes in the chest which otherwise escape detection. Moreover, the practise of artificial pneumothorax, which has lately been applied with such striking success in proper cases, could not have gained general acceptance but for skiagraphy.

The technic of  $x$ -ray examination, especially the comparative value of the various apparatus employed, will not be discussed here. This is the province of specially trained technicians. But every physician handling tuberculous cases should be able to read an  $x$ -ray plate and not depend entirely on the specialist radiographer for interpretation of the findings. When interpreted in connection with the clinical symptoms, with which the physician alone is acquainted, the  $x$ -rays yield the best results.

**Appearance of the Normal Chest.**—The appearance in the normal chest should be known before attempting to decipher pathological changes. It is, however, a fact that a normal chest, showing no signs suggestive of pathological conditions, is exceedingly rare. I have not yet seen one. Plate III shows a plate from a chest of a man apparently free from pulmonary disease.

While passing through the thorax, the rays are obstructed by the various tissues, according to their density, volume and constituent elements, and the result is that the denser tissues cast shadows on the screen or plate. The densest shadows seen are that of the heart and great vessels in the middle and to the left, and the diaphragm beneath.

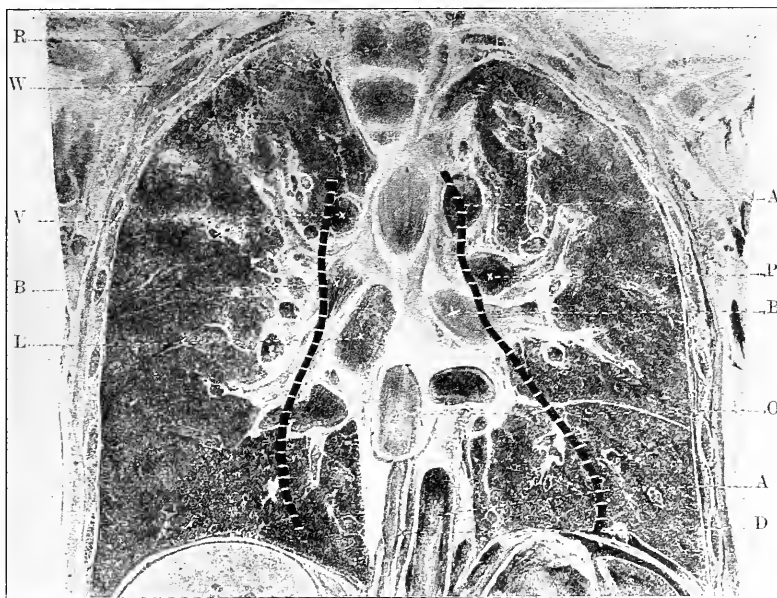


FIG. 64.—Structures making up the hilus shadow: *R*, second rib; *W*, second thoracic vertebra; *V*, arch of azygos vein; *B*, bronchus; *L*, bronchial lymphatic glands; *A*, aorta; *P*, pulmonary artery; *O*, esophagus; *D*, thoracic duct. (Doyen.)

Because it permits the rays to pass with less resistance than any other organ in the chest, the lung gives a dark image on the negative; the heart, the large vessels, the diaphragm and the liver, because of their density and blood content, obstruct the rays and produce light areas on the plate. The most translucent parts of the healthy viscera are the healthy lungs, but when they are collapsed by air in the pleura, as in pneumothorax, the space is even brighter. In healthy persons, when the patient takes a deep inspiration, the lungs brighten up. But the brightness of the lung tissue is not absolute. There is seen a delicate, at times even a more or less coarse, arborization, as of a network passing from the roots of the lung to the periphery. At the roots it

is caused by the greater density of the tissues, but in most persons also by the deposition of carbon particles, which may be found in nearly every individual over fifteen years of age. When the shadow at that point is abnormally accentuated, it may be an indication of enlargement or calcification of the glands, and in children it points to tuberculous tracheobronchial adenopathy. Often we note in this region small, sharply defined, oval opacities which represent optical sections of bloodvessels.

It is, however, difficult or impossible to evaluate every shadow or opacity because by their passage through the chest, the rays are obstructed by the various parts constituting the viscera, thus producing superimposed shadows. Carefully prepared stereoscopic pictures may enable us to distinguish these superimposed shadows in perspective, but they are after all not much superior to a good skiagram taken by instantaneous exposure. The excellent studies on the subject made in this country by Dunham, Boardman, Wolman,<sup>1</sup> Bibb and Gilliland,<sup>2</sup> and others have contributed considerably to our knowledge in this direction.

**The Hilus Shadow.**—The shadows seen at both sides of the heart are very frequently a source of confusion in diagnosis. As will be seen from Fig. 64 they are due to the density of the tissues composing the bronchi; the large vessels which are seen either in transverse or in optical section, combined with the opacities produced by the regional lymphatic glands and connective tissue, none of which can be differentiated on the screen or plate. While in some cases circumscribed opacities or spots represent calcified glands or nodules, in others they are produced by deposits of dust in the peribronchial lymphatic tissues which are very frequent in adults, and even in children in cities they are not uncommon. But in many cases simple engorgement of these tissues with blood is apt to give a shadow in that region. In fact during attacks of measles or whooping-cough the glands in the chest have been found visible in skiagraphic plates, and the same is often the case in acute affections of the respiratory tract in children or adults.

It is thus clear that many conditions, other than tuberculosis of the tracheobronchial glands, may cause shadows or opacities in the hilus region. Moreover, even when these opacities represent anthracotic or calcareous glands, the skiagram alone gives us no clue as to the activity of the process, which is after all the main problem in clinical diagnosis. In children it is hazardous to diagnose tracheobronchial adenopathy because of these opacities when the clinical picture is not in agreement.

To the right side of the heart the hilus shadow is more extensive than to the left because in the latter location the heart shadow obscures the hilus structures. In many cases we see strands passing from the

<sup>1</sup> Bull. Johns Hopkins Hosp., 1911, xxii, 229.

<sup>2</sup> Arch. Intern. Med., 1915, xv, 588.

hilus to the periphery or the diaphragm. It is the consensus of opinion that they are produced by bloodvessels and occasionally by bronchi which at times appear in optical section.

**Fluoroscopy.**—An  $x$ -ray examination of the chest should always be preceded by a fluoroscopic examination in a totally dark room. Inasmuch as this is very difficult to attain in the average physician's office, it is best done in the evening. With this we ascertain the shape of the thorax, the movements of the ribs and diaphragm, deformities of the bony thorax especially the spine, etc. In the healthy person the motion of the ribs is symmetrical. When the patient takes a deep breath, the lungs on both sides light up to the same degree. Unilateral limitation of motion of the ribs is suggestive of unilateral disease and phthisis is to be considered in this connection. When we find the ribs unduly horizontal, we should look for emphysema; when the horizontal setting is unilateral, pneumothorax is to be thought of.

Normally, the costal cartilages, especially in young subjects, are not distinctly visible in the radiogram. The ribs are sharply cut off (Fig. 2, Plate III). In older persons they are usually visible owing to ossification which takes place with advancing age. In tuberculous patients ossification of the costal cartilages, especially the first (Fig. 1, Plate VII), is very frequently seen on the skiagram. As was already stated Freund considers this a predisposing factor to phthisis because of the stenosis of the upper aperture of the thorax which it is apt to cause. In some cases of phthisis all the costal cartilages are calcified, and when examining a patient with the  $x$ -rays this point should not be neglected. But it must be mentioned that it is not an infallible sign of active phthisis. It may be found in persons who are not sick, while I have repeatedly found cases of advanced phthisis in which the costal cartilages were hardly visible.

The apices are carefully inspected, and the translucency of the lungs in these regions inquired into. Theoretically, it should be of equal intensity on both sides, but such perfection is only rarely encountered, even in healthy persons. Usually, owing to thickness of the muscles, scoliosis, etc., one side is somewhat darker. But this is best studied on the skiagraphic plate. With the fluoroscope we look for the "cough phenomenon," first described by Kreuzfuchs.<sup>1</sup> This author noted that in healthy individuals the translucency of the apices varies according to various conditions, especially the form of the chest. Deep respiratory efforts may clear up any shadow in healthy lungs. During cough the apices brighten up even when they are otherwise quite dark, excepting when there is diseased tissue in that region and the affected apex remains dark even during cough.

But this is not a very reliable sign. Jordan<sup>2</sup> says: "Failure of the apex to light up is difficult to make out with certainty; there are endless fallacies due to the position of the  $x$ -ray tube, the thickness

<sup>1</sup> Münch. med. Wehnschr., 1912, lix, 80.

<sup>2</sup> Lancet, 1914, i, 963.

of the pectoral muscles of the patient, the 'lie' of the ribs and clavicle, etc., and at best it is almost impossible to reproduce this 'failure' on a photographic plate with any certainty. I am quite sure that we should diagnose pulmonary tuberculosis in a large number of healthy subjects if we are to rely on this sign."

This view is shared by many, but it appears that Jordan is mistaken in his statement to the effect that the cough phenomenon cannot be reproduced on a skiagraphic plate. As will be noted on Plate V, F. Holst<sup>1</sup> has succeeded in reproducing this phenomenon very clearly. Moreover, this author has also shown that during cough there is an alteration in the lateral limits of the pulmonary apices, they become wider while the trachea becomes narrower, sometimes as much as 1 cm. In normal individuals this phenomenon is observed on both sides to the same degree, while in case one apex is altered by tuberculous changes, it fails to brighten up, and remains narrow and darker during cough. Of course this phenomenon is best studied on the screen, and only exceptionally may it be reproduced on a skiagraphic plate. We must, however, guard against mistaking the apparent changes in the brightness of the apices during cough caused by the separation of the ribs and widening of the intercostal spaces. It has been of immense service to me in many cases.

With the aid of fluoroscopy we also ascertain the size and position of the heart. In phthisis this organ is, as a rule, smaller than normal. In fact, when I find a large heart in a dubious case I hesitate before making a diagnosis of phthisis. In phthisis it is also very often vertical; it may be "hanging" cardioposis, and in more advanced cases frequently displaced toward the affected side.

After the apices, the diaphragm should claim our attention. The mobility of this muscle has been found defective on the affected side in many cases of phthisis; according to F. H. Williams,<sup>2</sup> in the very incipient stage. The motion of one-half of the diaphragm may not only be delayed when there is a pulmonary lesion, but it is at times seen to be "jerky," or "stammering," as Harold Mowat says. In some healthy persons the mobility of the diaphragm is very limited, while in most the breathing excursion is from three-fourths to one inch, and during forced respiration it may even move more than two inches, the left half of the muscle more than the right. When both sides are stationary, it may indicate emphysema, or nothing at all, but when one side moves while the other is immobile or its excursion is relatively limited, we should suspect tuberculosis. Various explanations have been given for this phenomenon. Some have attributed it to diminished power of retraction of the lung, others to implication of the terminal branches of the vagus, or of the phrenic nerve in the apical pleural thickenings, etc. In advanced cases limitation of motion may be due to pleural adhesions. It must

<sup>1</sup> Münch. med. Wehnschr., 1912, lix, 1659.

<sup>2</sup> Amer. Jour. Med. Sci., 1897, cxiv, 655.

however, be emphasized that in itself defective movement of the diaphragm may be found in healthy individuals. If unilateral it may be due to paresis of that muscle, or to an old basal pleurisy producing adhesions which hinder its excursion. In persons with big abdomens, the breathing is usually purely thoracic, and the diaphragm is immobile.

Extensive experience has shown limitation of motion on the affected side of the diaphragm in only a few cases of incipient phthisis. Indeed, we often see advanced cases in which both sides of the diaphragm are freely and equally mobile. On the other hand, limitation is found in non-tuberculous cases owing to adhesions remaining after previous attacks of pleurisy. In advanced cases this phenomenon has been considered in connection with the feasibility of artificial pneumothorax, but, as will be shown later on, it is not absolutely reliable.

In many cases we can diagnosticate diaphragmatic pleurisy by the marked elevation of the curve during inspiration; in others we note a series of small irregularities in the contour; in still others, bands of connective tissue are seen passing from the diaphragm to the lung.

**Skiagraphy.**—Of greater value in all stages of phthisis, especially in dubious early cases, is radiography. When properly taken and developed, the plate may be studied at leisure and slight alterations, which are not visible on the fluoroscopic screen, may be easily detected.

In evaluating the skiagraphic findings we must bear in mind the following points: Small infiltrations do not show any definite and clear-cut signs on the plate; at any rate, the shadow they cast is not pathognomonic. Cohn<sup>1</sup> inserted tuberculous tissue into healthy lungs of cadavers, of which he took radiograms and found that 1 c.c. of diseased tissue is not visible on the plate. Ziegler and Krause<sup>2</sup> have investigated the problem and found that pieces of tissue less bulky than 4 c.c. are not visible on the skiagram, and that, on the whole, small areas of infiltration are only visible when they are located near the surface of the lung. In other words, small infiltrations, when centrally located, are screened by normal pulmonary tissue, and may escape detection. When the lesion has caseated it casts a more or less dense shadow. But then the case is no more incipient.

In many cases we find that the affected apex is darker than its mate on the opposite side. In others, the affected area has the appearance of "ground glass." But even this does not invariably imply an active lesion. Indeed, it may be put down as a general rule that, in suspicious cases showing no constitutional symptoms, the darker the apex, the less likely the probability of its being a sign of *active incipient tuberculosis*. It may be revealing an old and healed lesion. I have been impressed with the following fact: A considerable proportion of apparently healthy people have one apex, usually the right, darker, due to various causes. In many it represents a healed tuberculous lesion, which is no longer serious. When in these individuals there

<sup>1</sup> Ztschr. f. Tuberkulose, 1911, xvii, 217.

<sup>2</sup> Röntgenatlas der Lungentuberkulose, Würzburg, 1910.

occurs a new tuberculous lesion in the opposite apex, which is not uncommon, it will give constitutional symptoms calling for a skiagraphic examination. The report from the radiographer may state that the lesion is located in the right side, while the physical signs show conclusively that the active lesion is in the left, or the reverse.

The divergence of findings on physical examination and skiagraphy is best seen in far-advanced cases of phthisis in which a new lesion occurs in the hitherto unaffected apex. The plate does not show it until caseation has taken place, while physical exploration reveals it clearly. I have had this incontrovertible proof of the inadequacy of skiagraphy in incipient lesions repeatedly.

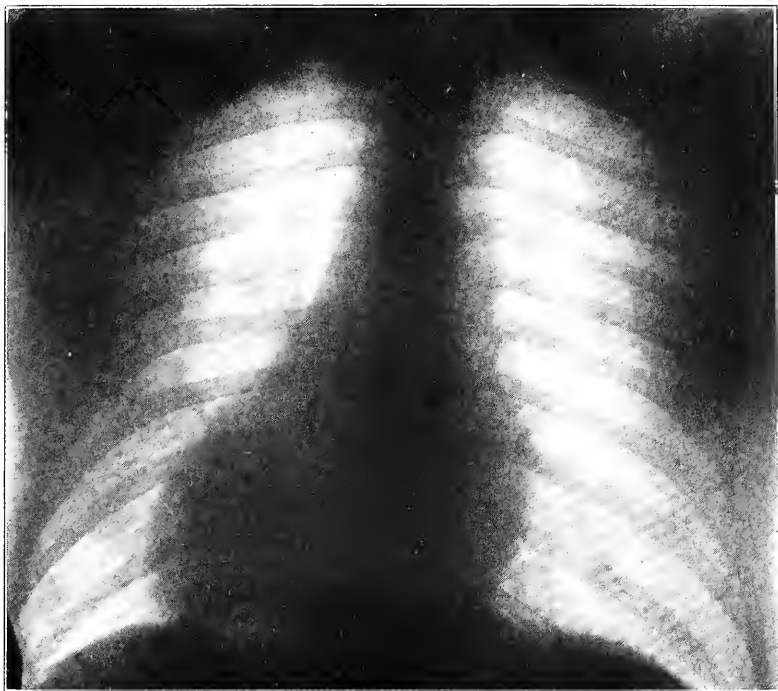
For these reasons we should not conclude merely on finding opacities in one apex that we are dealing with a case of incipient phthisis. When found in connection with constitutional symptoms and signs on physical exploration these opacities are of diagnostic value. Nor should we conclude in the presence of constitutional symptoms and local signs suggestive of phthisis, but negative skiagraphic findings, that a case is not tuberculous. Such a case requires further observation, despite the negative *x*-ray findings. I do not hesitate to make a diagnosis of pulmonary tuberculosis under such circumstances when clinical evidence warrants it.

After the apex we carefully examine the condition of the roots of the lungs, the hilus, with a view of ascertaining the presence of enlarged caseated or calcified glands, or peribronchial infiltrations in that region. The shadows and mottlings observable at these points have been discussed. At first there was a tendency to consider all abnormalities as evidences of enlarged glands and a diagnosis of tuberculosis or tuberculous adenopathy was made on this evidence alone. But experience has shown conclusively that this shadow may be caused by any congestive condition of the bronchi and lungs, and it is not pathognomonic of phthisis. There is hardly an adult living in a city, or working at a dusty trade who has no peribronchial thickening, enlarged or calcified glands at the hilus of the lungs. It was also found by Cohn, Dunham, Boardman, Wolman, Bibb and Gilliland, and others, that, excepting in cases with calcified glands, these shadows are caused by blood in the vessels of the thorax. Blood absorbs the *x*-rays more readily than infiltrated soft tissue or sputum. Experimental injection of the arteries in the lungs intensifies the shadow, and in human beings injection of the vessels with substances giving a strong shadow, produce pictures which are exactly like those of normal lung markings.

This fact explains many of the thickenings and strands noted on chest plates, running from the hilus to the periphery of the lungs. In some cases they are due to bronchitis with congestion; in others the mottling is due to calcified glands which are harmless and of no clinical importance. Sewall and Childs report the case of a presumably non-tuberculous stone-cutter furnishing a skiagram in which,

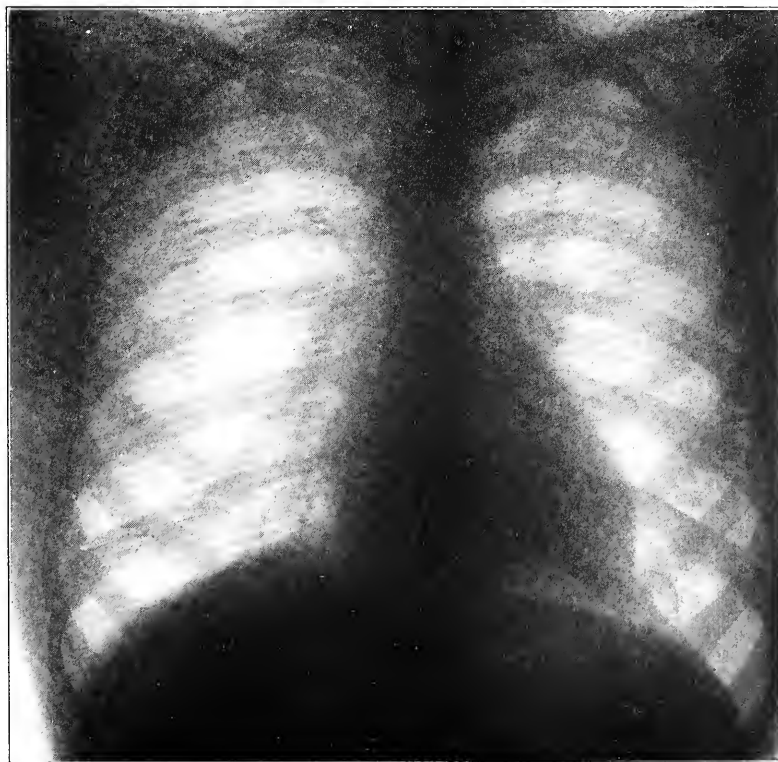


FIG. 1



Radiogram of a man with apparently healthy thoracic viscera. Dorsoventral position.

FIG. 2

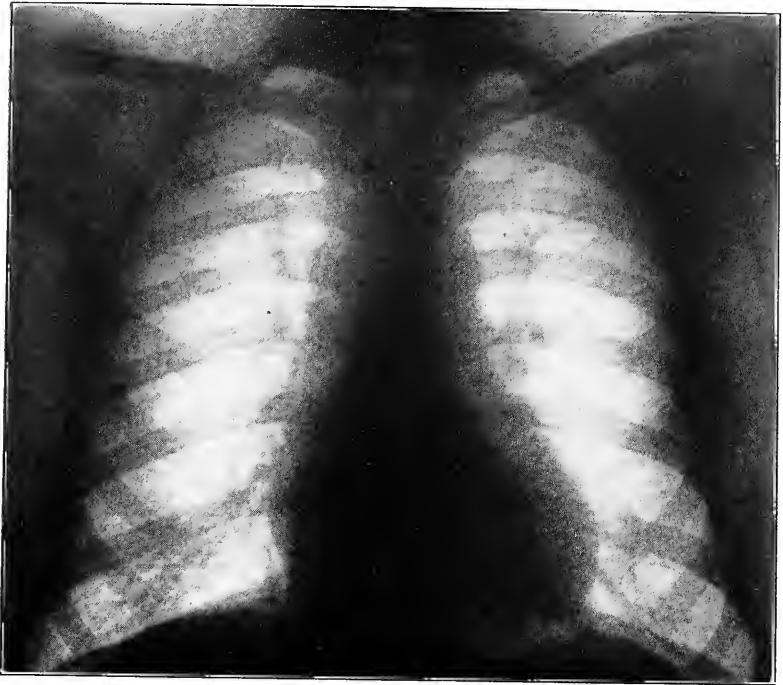


Same man as in Fig. 1, but in the ventrodorsal position.



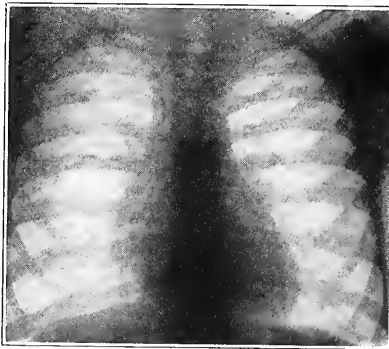
PLATE IV

FIG. 1



Radiogram of a woman with apparently healthy thoracic viscera.

FIG. 2



Radiogram of the chest of a child eight years old. Though no symptoms or signs of tracheobronchial adenopathy could be found clinically, the radiogram shows shadows suggestive of such a condition.



PLATE V

FIG. 1



Lung apex during ordinary breathing.

FIG. 2



Apex during ordinary breathing.

FIG 3



The same apex while patient is coughing, and showing a narrowing of the trachea, widening, and lightening up of the apices, especially the right. (F. Holst.)

FIG. 4



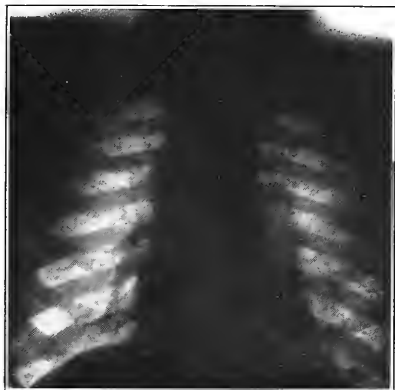
The same apex while patient is coughing, showing narrowing of the trachea, and lightening up of the area of the lung. (F. Holst.)

The "Cough Phenomenon."



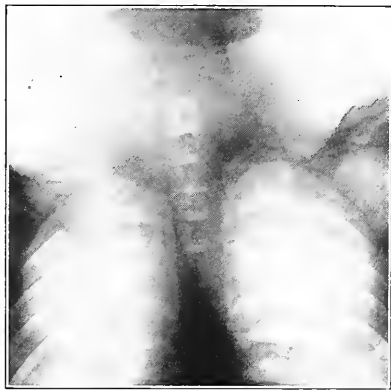
## PLATE VI

FIG. 1



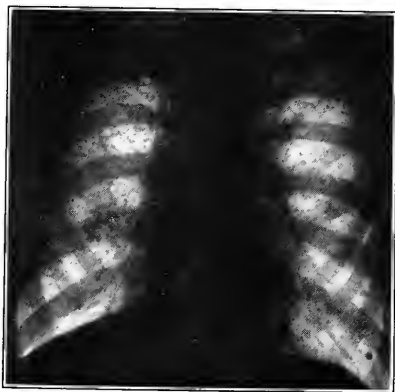
Radiogram of a case of abortive tuberculosis. Though suggestive of an extensive lesion in the left apex, the physical signs, as well as the course of the disease, showed that the activity of the process was benign. The patient recovered within three months.

FIG. 2



Radiogram of the apices in a case of incipient phthisis. No definite changes are visible, though physical exploration revealed a distinct lesion in the left apex, and the constitutional symptoms were clearly those of phthisis.

FIG. 3



Slight infiltration of the right apex. Marked increase in lymphatic tissue in both hilus regions.

FIG. 4



Partial consolidation of both apices, large cavity in left apex. Dilatation of bronchi of lower lobe of left lung. Heart displaced to the left.





## PLATE VII

FIG. 1



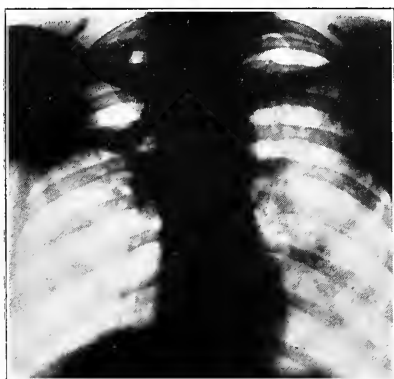
Infiltration of right apex. Peribronchial infiltrations and calcified glands at the hilus on both sides.

FIG. 2



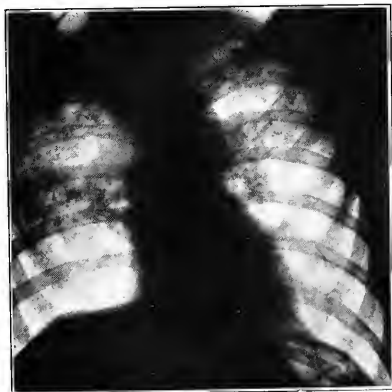
Very dense infiltration of right upper lobe and large cavity below the clavicle. Marked peribronchial infiltrations. The hilus region on both sides shows increase in lymphatic tissue.

FIG. 3



Large cavity surrounded by a dense fibrous wall in upper part of right lung. Enlarged glands in right hilus region. Lower half emphysematous. Left lung shows moderate infiltration beneath the clavicle and enlarged hilus glands. Drop heart.

FIG. 4

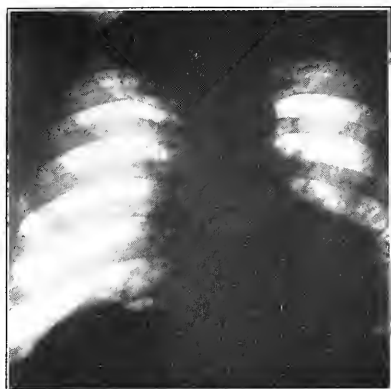


Bilateral tuberculous infiltration of both lungs. Dense hilus region due to calcification of glands. Several small cavities in right lung. Adhesions of diaphragm. Trachea markedly pulled over to the right. Stomach visible at left base.



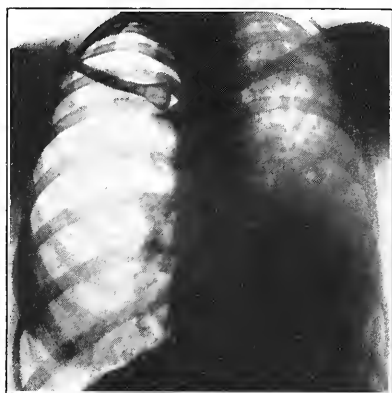
## PLATE VIII

FIG. 1



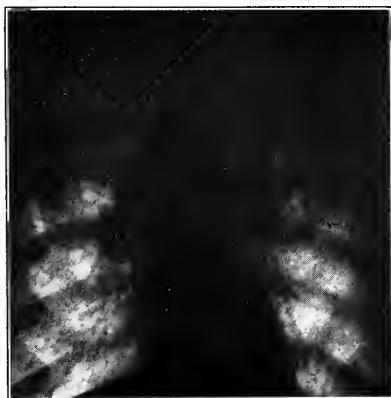
Slight infiltration of both apices. Coarse infiltration of lower half of left lung with thickened pleura. Heart pulled over to the left and downward. Emphysema of right lung. Diaphragm in right side shows a bulging due to adhesions.

FIG. 2



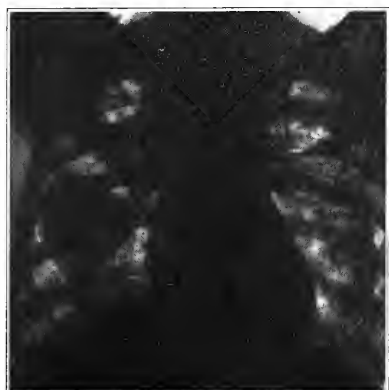
Dense infiltration of upper third of left lung. The rest presents a dense homogeneous shadow caused by consolidation of pulmonary parenchyma as well as thickened pleura. Right lung emphysematous and several enlarged and calcified glands are seen at the hilus.

FIG. 3



Dense infiltrations of both apices. Miliary-like infiltrations through both lungs. Hilus glands greatly enlarged and apparently calcified. Trachea pulled over to the right. Heart small and dropped; aorta dilated.

FIG. 4



Diffuse nodular infiltration of both lungs with multiple cavitation.



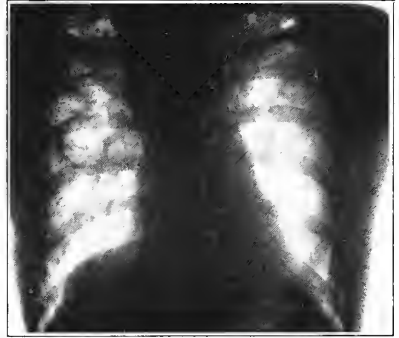
## PLATE IX

FIG. 1



Dense infiltration of lower half of right lung with thickened pleura. Large cavity in left lung occupying apex on a level with first two interspaces. Drop heart.

FIG. 2



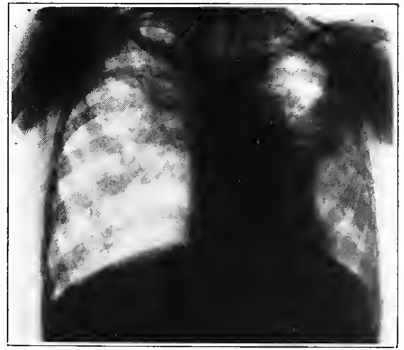
Diffuse infiltration of both lung apices. Round cavity, surrounded by a dense fibrous capsule, under the right third interspace in mammillary line. Irregularity of the diaphragm due to adhesions.

FIG. 3



Large, oval-shaped cavity in right apex. Lymphatic tissue at hilus increased. Cavity in middle portion of left lung at third interspace. Heart dropped; pleuro-pericardial adhesions.

FIG. 4



Chronic cavitary phthisis in a child eight years of age, with displacement of the heart to the left.



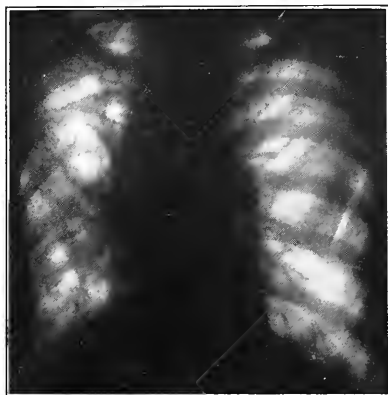
## PLATE X

FIG. 1



Syphilis of the lung simulating in the radiogram a tuberculous lesion in the right apex.

FIG. 2



Pulmonary Syphilis. Diffuse peribronchial infiltrations of right lung, mostly marked at the lower half. Hilus glands in left lung are distinctly enlarged. Pericardial adhesions mainly seen in right side.

FIG. 3



Radiogram of a child nine years old, suggestive of enlarged hilus glands. The symptoms and signs of this disease were, however, lacking. Yet on a level with the second rib an opacity suggestive of a a calcified gland can be seen.





## PLATE XI

FIG. 1

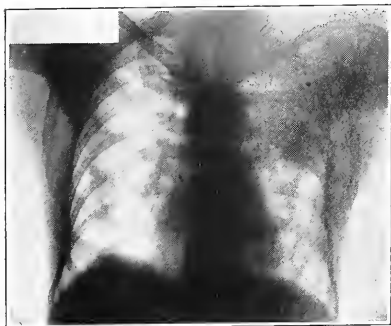


FIG. 2

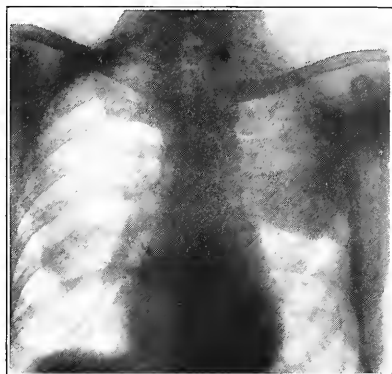
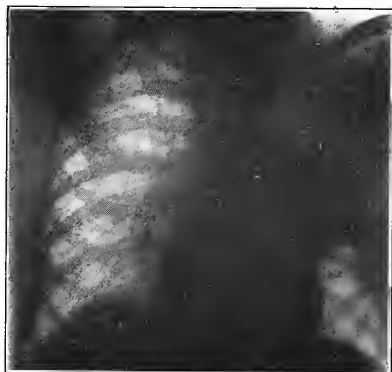


FIG. 3



FIG. 4



Malignant tumor of the left lung. In the first radiogram the shadow could not be differentiated from a tuberculous lesion. It was only in the third radiogram, taken three months later, that the true nature of the affection could be made out radiographically.



except for the relatively moderate involvement of the apices, the mineral deposits occasioned opacities resembling the densest tuberculous structure. I have often had the same experience with workers at dusty trades. The criterion given by some authors for distinguishing inactive consolidations and calcified glands from shadows representing active lesions by the fact that the latter appear "wooly," does not hold in many cases. Any structure out of focus appears diffuse—"wooly"; even instantaneously taken plates are not free from this source of error. "The interpretation of less dense and more diffuse opacities is chiefly guesswork" say Sewall and Childs.<sup>1</sup> "They usually represent either pathological lymph nodes or bloodvessels in more or less optical section."

**Sources of Error.**—The analysis of these shadows and mottlings admits of so many interpretations, that they are of doubtful utility in most incipient cases. The "ground-glass" appearance of an apex is found in plates taken from healthy individuals. A shadow, when not the result of scoliosis, shows that there is some airless tissue in that location. But we are not justified in invariably assuming that it was caused by a tuberculous infiltration; or even if so, that the lesion is active. Ziegler and Krause, Dehn, Arnsperger and others have found that calcified and caseated tissue, and even fluid, anthracotic and calcified lymph glands produce the same radiographic shadows. I have seen a large empyema failing to disclose itself on an *x*-ray plate.

There is no more justification for placing an individual, one of whose apices casts a shadow on a plate, under prolonged and costly treatment than there is for the treatment of one for mitral insufficiency merely because he has a systolic murmur at the cardiac apex. In both cases the clinical symptoms decide whether the person is sick and in need of treatment.

Because we are looking, in incipient cases, for small areas of recent infiltration, it is clear that we cannot rely on skiagraphy alone for the diagnosis of early phthisis. The skiagraphic picture gives the history of the thoracic viscera throughout the life of their owner. Any pathological change which may have occurred at any time may have left traces behind which are likely to cast shadows or cause opacities on the plate. For this reason, in incipient or dubious cases the skiagraphic findings are to be taken only in connection with constitutional symptoms and physical exploration of the chest. If the latter are negative, the case is to be considered non-tuberculous, no matter what the skiagraphic plate shows.

It is thus clear that in the diagnosis of incipient phthisis the *x*-rays are not of the value which some authors have attributed to them. Early tuberculous lesions, slightly enlarged bronchial glands, unless caseated or calcified, as well as mucous secretions, usually permit the rays to pass through without casting any shadows on the plate.

<sup>1</sup> Arch. Intern. Medicine, 1912, x, 45.

Optical sections of bloodvessels, due to any condition that may cause vascular engorgement, may show opacities on the plate simulating the characteristics of tuberculous lesions and may lead to error.

What is of most importance in obscure lesions is not so much their causation, but their activity. A healed tuberculous lesion in an apex is not incompatible with excellent health, as was repeatedly emphasized. But it produces a shadow on the skiagram as well as, often better than, an active lesion.

Skiagraphy may be of great assistance in attempts at localization of a lesion, though smaller tuberculous foci may often be discovered with the orthodox clinical methods of diagnosis and the determination of the activity of an apical process can only be accomplished by careful observation of the case, paying special attention to the constitutional symptoms, such as the temperature, the pulse, cough, expectoration, and the physical signs. "With our present ability to produce and interpret *x*-ray pictures," say Sewall and Childs, "it must be admitted that a judgment founded on clinical history combined with physical signs may lead to a strong suspicion of tuberculous infection long before any signs of actual tissue changes, except those involving bronchial glands, appear on the *x*-ray negative." Wolman,<sup>1</sup> who has worked with the stereograph, arrives at a similar conclusion. He says: "In the great bulk of cases the stereograph tells us no more than a careful clinical examination, yet in a fair number of cases, and those among the most interesting and puzzling, it gives additional information. But we must add the caution that a careful history is indispensable, since not even the stereograph can tell an active from a healed lesion."

**Skiagraphy in Advanced Stages of Phthisis.**—In my experience skiagraphy has been of greater utility in the diagnosis of advanced disease than in early or dubious cases. Very often we find that the *x*-ray plate reveals more extensive involvement than the findings on physical exploration of the chest, and the prognostic significance is thus invaluable. In cases in which the question of artificial pneumothorax is considered, skiagraphy offers invaluable assistance. Very often pleural effusions, especially the localized or interlobar varieties, are discovered, though they have escaped detection by routine methods. The same is true of localized pneumothorax.

The radiographic picture of advanced phthisis is variegated, depending on the changes in the lungs and pleura. The intensity of the shadows cast by the lesions depends on their nature and density. Caseated and calcified areas cast dense shadows, while proliferation of tissue, especially when it is also congested, or fibrosis is also clearly detected. Old, indurated areas are usually more or less sharply demarcated from the surrounding tissues, while with new, active infiltrations the shadow merges by degrees with the surrounding air-

<sup>1</sup> Johns Hopkins Hosp. Bull., 1911, xxii, 236.

containing lung tissue. Thick pleura is discovered by a dense, uniform shadow, and all connective-tissue formations reveal themselves in the same manner. More often than by physical exploration, cavities disclose themselves by showing limited areas lacking in lung markings and surrounded by thick shadows (Plate VII). They may often be seen moving during inspiration and expiration when examined with the fluoroscopic screen. But when a cavity is filled with secretions, it is again airless, and casts the shadow of the surrounding tissues, and a very much thickened pleura may cover up a cavity. A cavity may also be screened by the surrounding healthy lung tissue. Thus, we often fail to find it with the  $x$ -rays, while physical exploration reveals it easily. "One must use great caution in diagnosing cavities as the result of the plate alone," says Ray W. Matson. "Adhesive bands when circularly arranged, so closely resemble cavities that even an expert will make mistakes if his work is not controlled by physical examination and clinical history."

Kuthy and Wolff-Eisner point out a fact which in my experience occurs very often: When the signs found by percussion show a more extensive lesion than the radiogram shows, then it is the thickened pleura which produces the dulness. Conversely, when the signs obtained by percussion are of smaller extent than the radiogram reveals, there is a central parenchymatous lesion of very serious import.

The condition of the pleura may be studied on the plate. Fibrinous pleurisy is not shown at all. But effusions reveal themselves clearly as an intense shadow on the plate. Its upper level is clearly demarcated from the lung above, and in the fluoroscope it may be seen moving somewhat with the respiratory movements. When the quantity of fluid is small, it may escape detection when sinking down in the diaphragm. In hydropneumothorax it is important that the exposure should be made with the patient in the erect posture, because when lying down small quantities of fluid spread in a thin layer and may escape detection. In hydropneumothorax the upper layer of the fluid forms a sharp line, while in pleurisy with effusion the upper level is usually not so sharp, but gradually merges with the lung tissue above it. The fact that in the latter case the level does not shift with motion of the patient's chest, shows that it is not a hydropneumothorax; in the latter case it does shift (see Plate XVI, Fig. 2).

The displacements of the mediastinum caused by pleural effusion are best made out with the  $x$ -rays; but it is impossible to distinguish between fluid and the liver in right-sided effusions. Dislocation of the trachea and larynx may often be discovered on the plate (Plate VIII).

**Skiagraphy in Pneumothorax and Pulmonary Emphysema.**—Skiagraphy finds its greatest field of usefulness in our attempts at discerning the changes in the thoracic organs during the course of phthisis in revealing pneumothorax, especially of the localized variety which formerly escaped recognition. Complete pneumothorax appears

clearly on the fluorescent screen or the skiagraphic plate as a bright area, lacking in lung markings; in contrast with the opposite expanded lung it may be said to be brilliant. The collapsed lung is seen lying near the mediastinum or against the spinal column as a dark band. Deep breathing has no influence in changing the appearance of the affected area. The dome of the diaphragm is lower than, and the mediastinum is displaced to, the unaffected side. The forms of pneumothorax which are localized, "silent," and may even give no symptoms, or only indefinite symptoms and signs, are easily discerned. In those anomalous cases in which pleural adhesions prevent the induction of an artificial pneumothorax, still negative pressure is obtained and gas is inflated, we could not be enlightened that our labors are vain efforts without skiagraphy showing that no pneumothorax has been created despite the fact that several inflations of gas have been made. A pneumothorax occupying a part of the chest, as is shown in Plate XIV, Fig. 3, could not be discovered without the aid of skiagraphy.

Emphysema, too, is easily recognized with the  $x$ -rays. The lungs are brighter than normal because of the dilated alveoli and greater air content. But the translucency is not as intense as in pneumothorax, and lung markings are still made out. The ribs are widely separated, run horizontally, and their motion during respiration is impaired. During the course of phthisis the unaffected lung is often found emphysematous owing to vicarious function; often we find only parts of one lung, or one lobe emphysematous for this reason. Skiagraphy clears these matters up.

## CHAPTER XVIII.

### THE CLINICAL FORMS OF PHTHISIS.

#### POLYMORPHISM OF THE CLINICAL PHENOMENA OF PHTHISIS.

LAENNEC showed clearly the unity of the elemental pathological changes found in phthisis and Koch, discovering the tubercle bacillus, proved it etiologically. But all attempts to impose this unity on the clinical manifestations of tuberculous diseases of the lungs have failed dismally. In pathology, particularly in clinical medicine, unity of causation does not always indicate unity of effect. Especially is this true of a polymorphous disease, as pulmonary phthisis.

A study of the morbid anatomy of phthisis shows great polymorphism—there are hardly two cases showing the same changes in structure. There are cases in which the lesions are purely proliferative, characterized by the formation of tubercles, as is the case with acute miliary tuberculosis; in others they are mainly exudative, as in chronic phthisis. But in the latter the difference in the intensity of the productive inflammation, which tends to limit the morbid process, and the process of necrosis, which tends to extend it, produce a diversity of lesions which have important bearings on the clinical picture, course, and prognosis of the disease.

This is to be expected when we consider that the disease produced by the tubercle bacilli depends on the interaction of two forces of inconstant intensity, viz.:

1. On the intensity of the infection. This depends on the number of bacilli which have entered the body; their virulence which we know is variable, depending on the type, and the condition under which they existed before entering the body, etc., and on the portals of entry. It is doubtful whether infection by inhalation will produce the same clinical picture as infection by ingestion or by inoculation; whether hematogenic tuberculosis will produce the same symptoms as aërogenic or lymphogenic infection.

2. On the resistance of the host, which is also an inconstant value, depending as it does on certain and uncertain, constant and temporary, conditions which cannot always be defined clearly. Thus, the effects of the infection depend on the age at which it has taken place. During the first six or twelve months of life massive infection produces a different disease than during the succeeding years of childhood. Acute miliary tuberculosis is common at the former age, while tuberculosis of the glands, bones and joints is mostly seen at the later ages. Primary infection of an adult is usually followed by clinical

phenomena which differ markedly from those seen in individuals who were presumably infected during childhood and the bacilli remained dormant for many years. We have already discussed the effects of preëxisting diseases on the type and course of phthisis.

"To speak of pulmonary tuberculosis as an entity," says von Hanse-  
mann,<sup>1</sup> "and to describe it as one disease caused by the tubercle bacillus is hardly conceivable. One has to compare pure miliary tuberculosis of the lungs with chronic indurative phthisis, and the latter with acute florid phthisis or caseous hepatization of the lungs, to find clearly that they are different pathological pictures which defy all comparisons. For these reasons it is altogether impossible to speak simply of pulmonary tuberculosis and thereby retain a clear survey of the different forms of the disease. In reality we are compelled to draw a sharp line of demarcation between these different forms of the disease, even when we are inclined to consider the tubercle bacilli as the underlying etiological cause of all the forms of the disease."

**The Stages of Phthisis.**—Early writers on phthisis, who were innocent of modern methods of diagnosis, felt constrained to differentiate various forms of the disease as they saw it clinically. They divided it into three stages: *Phthisis incipiens*, *phthisis confirmata*, and *phthisis desperata*. Bayle, in the first decenium of the nineteenth century, added a fourth stage, *Phthisis occulta*, or *germe de la phthisie*, which corresponds to the modern pretuberculous stage, when the tubercles in the lung are too few to produce symptoms. Laennec, who was an excellent and pioneer pathologist and clinician, having invented auscultation, divided phthisis into three stages, basing his classification on anatomical grounds. He divided phthisis into: *First stage*, the accumulation of the tubercles, which betray themselves by bronchophony and dulness over the affected area; *second stage*, softening of the lesion, producing bronchial breathing, coarse rales and pectoriloquy; and *third stage*, the elimination of the softened area, leaving pulmonary excavations which may be found by careful physical exploration.

This division of phthisis into three or four stages has remained to date not only among the laity, who fear the second and third stages, but also among physicians, who are always aiming at discovering the disease in the pretuberculous stage, or at least in the first, or incipient stage. Some even maintain that the disease is curable only at this stage. That this is not always true will be shown later on.

#### OFFICIAL CLASSIFICATIONS OF THE STAGES OF PHTHISIS.

With the advance of knowledge of the clinical manifestations, and the methods of recognition of the disease, the stages into which phthisis is divided remained practically the same. They have only been more

<sup>1</sup> Berl. klin. Wehnschr., 1911, xlvii, 1.



exactly defined. In Germany the classifications of Turban and Gerhardt have gained wide acceptance, while in this country, the American Sanatorium Association and the National Association for the Study and Prevention of Tuberculosis have adapted the following classification:

**Incipient.**—Slight initial lesion in the form of infiltration limited to the apex of one or both lungs or a small part of one lobe. No tuberculous complications. Slight or no constitutional symptoms (particularly including gastric or intestinal disturbance or rapid loss of weight).

Expectoration usually small in amount or absent.

Tubercle bacilli may be present or absent.

**Moderately Advanced.**—No marked impairment of function, either local or constitutional. Localized consolidation moderate in extent with little or no evidence of destruction of tissue or disseminated fibroid deposits. No serious complications.

**Far Advanced.**—Marked impairment of function, local and constitutional. Localized consolidation intense, or disseminated areas of softening, or serious complications.

**Shortcomings of the Official Classifications.**—If the object of this classification is to define the prognosis of phthisis, it fails utterly. A patient with a "slight initial lesion in the form of an infiltration of the apex" has not always a greater expectation of life than one having "marked local impairment of function, and extensive destruction of tissue." In fact, in acute miliary tuberculosis of the lungs, the lesion is so slight that it can often not be localized during life. On the other hand, many cases of phthisis with extensive excavations have a better outlook, at least as regards duration of life, and even as regards regaining efficiency, than some with limited lesions at one apex, without expectoration of tubercle bacilli but with evidences of toxic activity. Moreover, it is clinically wrong to put into one class the incipient cases showing no fever, no tachycardia "at any time during the twenty-four hours," no gastric or intestinal disturbances, no rapid loss of weight, etc., which are evidently cases of abortive tuberculosis, if at all actively tuberculous, with those having lesions limited to one or both sides and who do show constitutional symptoms of toxemia. The former will recover within a few months under any rational form of treatment, or spontaneously, while the latter may not, even with the most rigid institutional, climatic, dietetic, or specific treatment.

Any physician having opportunities to observe many tuberculous cases is struck with the fact that the prognosis, immediate and ultimate, does not entirely depend on the changes in the breath sounds, the presence or absence of rales and signs of excavations in the lungs. The constitutional symptoms such as fever, pulse rate, presence or absence of dyspnea, gastric disturbances, and above all the resistance of the patient, play a greater role in the ultimate outcome of a case than the anatomical changes.

In order that a case may be considered "incipient," according to this classification, and nearly all others which have been devised, the constitutional disturbances must be slight or absent. Thus, in the definition of terms it is stated that "the impairment of health may be so slight that the patient does not look or feel sick in the ordinary sense of the word." The pulse should not exceed 90 per minute and the temperature 99.5° F., and the sputum may be negative. The physical signs consist in "slight prominence of the clavicle, lessened movement of the chest, narrowing of the apical resonance with lessened movement of the base of the lung, slight, or no change in resonance, distinct or loud and harsh breathing with or without some changes in the rhythm (*i. e.*, prolonged expiration), vocal resonance possibly slightly increased; or fine or moderately coarse rales present or absent. If sputum contains tubercle bacilli, any one of these." Considering that the apex is defined as "that portion of the lung situated above the clavicle and the third vertebral spine," it is clear that the lesion must be quite limited, often of the type considered "dubious" by some clinicians.

All these symptoms, or absence of constitutional symptoms, and signs in the chest may be found in a large proportion of persons in all walks of life, working hard at their occupations, who, if followed for many years, are not found to develop active phthisis. People with collapse induration often show more distinct physical signs at one apex, yet they are not phthisical.

On the other hand, a really phthisical person showing so few signs on physical exploration, but in whom the disease is pursuing an acute or subacute course, may be carried off much quicker than many with extensive involvement, but manifesting a tendency to chronicity of the process.

It cannot be denied that these three or four stages of tuberculosis are altogether arbitrary. We cannot often separate them by sharp lines of demarcation and say "this is a first stage case," or "this case is passing from the second into the third stage," etc. There are always transitional forms. There are also numerous cases showing healed lesions which at the time of activity were in the third stage, but give no more trouble—while an initial lesion in the other lung is responsible for the disease for which the patient consults the physician. Such cases, incipient in the true sense of the word, must be considered far advanced according to this classification. It is also a fact that, for phthisis to end fatally, it is not necessary that the lesion in the lung should soften and produce a cavity; caseation alone, when extending rapidly, may kill; the patient has thus not reached the third stage, yet he dies. On the other hand, we have numerous patients, who, despite the fact that they have more or less extensive excavations in the lungs, are in fact in the third stage of phthisis, yet they feel well, and are even efficient at their occupations, and when they finally die the cause may be another disease.

For these reasons some clinicians have been constrained to distinguish the various forms of phthisis met with in practice into different clinical entities. Thus, even the classification mentioned above considers acute miliary tuberculosis as a distinct disease. Other authors, like Alfred Loomis, Williams, Andrew Clark, Douglas Powell, etc., have described fibroid phthisis—which in the above classification would always be included among the advanced cases—as a distinct disease.

Many writers on this subject have gone much further and distinguished not only acute and chronic forms of the disease but have also described congenital, or hereditary and acquired, forms of the disease; phthisis in arthritic, gouty, diabetic, nephritic, alcoholic, or syphilitic subjects; also according to some prominent symptoms, such as hemorrhagic, bronchitic, bronchiectatic, pleuritic phthisis. In accordance with certain etiological factors, there has been described phthisis in workers at dusty occupations, such as miner's phthisis, etc. Finally tuberculosis of the lungs in children has always been considered as presenting a different clinical picture from that in the adult; while in aged persons the symptomatology of phthisis differs from that in younger individuals.

**Classification in the Present Work.**—The classification of the diversity of clinical types of tuberculosis of the lungs, to be of practical value, if it is to be attempted at all, must have a prognostic value. For this reason the acute forms of the disease are to be separated into a class by themselves, as has, in fact, been done by all writers on the subject. In chronic phthisis the ultimate outcome of the disease depends mainly on the relative intensity of the two processes in the lungs, the destructive and the reparative, the former manifesting itself by caseation and softening, and the latter by the formation of fibrous tissue which limits the destructive process and even heals the lesion by cicatrization. Both processes, fibrosis and necrosis, are caused by the tubercle bacilli. And inasmuch as there are many cases in which the fibrosis dominates the anatomical changes in the lungs, and the symptoms thus produced differ from those in which the caseating process predominates, it is clear that there is justification for separation of fibroid phthisis into a distinct class of the disease. This justification is fortified by the fact that the prognosis of fibroid phthisis is distinctly more favorable than that of chronic caseous phthisis, and the treatment indicated is different than that in other forms.

In common chronic phthisis we find that among the cases which have been described as "incipient," there are many which show a marked tendency to cicatrization of the lesion, spontaneously or after some treatment for a few months. In the vast majority of cases this form of phthisis is not at all recognized and only at the autopsy some scars or calcified foci are found in the lung or pleura showing that the person had survived a tuberculous lesion. To treat these cases in the same manner as we treat common chronic phthisis is

wrong. We should, when diagnosticating a case of this kind, tell the patient that his malady is relatively trifling, and that he will recover within a few months, if he observes ordinary hygienic and dietetic rules. We can often also spare him the trouble and the economic danger of giving up his business which is usually necessary in cases of chronic phthisis. We have therefore described abortive tuberculosis as a distinct clinical type of the disease.

Most of the victims of tuberculosis who succumb to the disease, or who suffer from it for long periods of time even if they recover, are affected with chronic phthisis. This disease is characterized by an undulating course, marked by periods of quiescence of longer or shorter duration, and interrupted by periods of acute or subacute exacerbations. In fact it may be stated that acute progressive phthisis, or galloping consumption, consists clinically in an acute exacerbation of the disease which is not followed by a period of quiescence. In the chronic type of the disease, proper and timely treatment may save the patient, while negligence in this regard is apt to prove disastrous. For this reason it is imperative that it should be recognized as early as possible. We have therefore divided the subject into two parts: incipient phthisis and advanced phthisis. The former, if recognized in time, and appropriate treatment applied, may often be aborted; or acute exacerbations leading to irreparable damage of the lungs and other organs and functions may be prevented. The latter, when properly cared for, may be kept in check so that acute exacerbations occur less frequently, or not at all, and cicatrization of the lesion goes on unhindered.

We also know that tuberculosis in children is anatomically, and also clinically, not of the same character as that in adults. In the former the glands, bones and joints, while in the latter the lungs, are mainly the organs which bear the brunt of the infection. Indeed, considerable harm is done to children by treating them for chronic pulmonary tuberculosis which, before the eighth year of life, they practically never have. For this reason, the disease as it occurs in infants and children merits separate description. Because in infancy the infection is usually followed by acute manifestations, while in children between two and ten years of age chronic disease of the glands occurs, we shall speak of tuberculosis in infants, and tuberculosis in children.

Finally, it is now known that phthisis occurs in the aged just as frequently as in younger individuals, but that it is not recognized very often because of the peculiar symptomatology it presents. The aged consumptives, believing that they only suffer from chronic bronchitis, asthma or pulmonary emphysema, are sources of infection to an extent not as fully appreciated as they deserve. We have therefore devoted a special chapter dealing with tuberculosis in the aged, pointing out its clinical characterization.

These forms of phthisis do not exhaust the subject of the clinical polymorphism of this disease. There are many other types which

may be appreciated when carefully studying the cases, while quite often these types overlap one another to an extent as to render it difficult to decide to which class a case belongs. But for practical purposes these clinical classes are sufficient. They assist in appreciating the course of the disease when it occurs, and give us hints for prognosis and treatment which are invaluable and which cannot be had when pulmonary tuberculosis is considered as a single clinical entity.

We shall therefore describe phthisis under the following headings:

1. Chronic phthisis, incipient stage.
2. Chronic phthisis, advanced stage.
3. Acute phthisis.
4. Fibroid phthisis.
5. Abortive pulmonary tuberculosis.
6. Pulmonary tuberculosis in children.
7. Phthisis in the aged.

## CHAPTER XIX.

### CHRONIC PHTHISIS. INCIPIENT STAGE.

#### INCIPIENT PHTHISIS.

**Onset.**—A lay writer,<sup>1</sup> describing his own, subsequently fatal case of phthisis, in speaking of his "initiation into T. B.," says: "The entrances are innumerable, however sole the exit. Indeed, the initiation varies so widely that one would not be far wrong in saying that it is never twice the same. Yet many initiations have certain features in common; and in a general way it may be said that all belong to one of two great classes—the sudden and the protracted." No physician, however extensive his experience with phthisis, could do more justice to the subject, or make a better generalization of the various ways in which phthisis is likely to begin.

A sudden or abrupt onset of phthisis is infrequent, but it does occur. We meet with patients who have been in the best of health; have no ascertainable hereditary taint; have not come into immediate or intimate contact with a consumptive as far as they can remember; have not overworked, not suffered from exposure, but they suddenly begin to cough, lose weight, have fever, feel tired at the least exertion, and a careful physical examination reveals a small, but progressive lesion at one apex. We meet with others who, without any premonitory symptoms, without any exciting cause, suddenly perceive a warm sensation in the throat, cough and bring up a mouthful of blood. The hemoptysis may be scanty or copious, but the signs elicited while examining the chest leave no doubt that it is derived from a pulmonary lesion, and the subsequent course of the disease proves conclusively that we are dealing with phthisis. Still others, after an indiscreet exposure to the vicissitudes of the weather, or after a cold bath to which they are not accustomed, begin to cough and are treated for a "cold," "grippe," etc., for some time. But the symptoms fail to ameliorate in spite of careful treatment, when one day a careful examination of the chest shows a distinct lesion, or a bacteriological examination of the sputum reveals the presence of tubercle bacilli. In some, exposure may bring on an attack of pleurisy, dry or with effusion, the subsequent course of which is distinctly that of phthisis.

But in a large proportion of cases the disease develops slowly, insidiously—the initiation is protracted as our lay friend said. For months or years the patient has not been well. He was "subject to colds," and every autumn or winter he passed through one or more attacks

<sup>1</sup> The Atlantic Monthly, June, 1914, cxiii, 747.

of "grippe," bronchitis, etc., with cough, expectoration, fever, malaise, etc., but he soon recuperated and worked more or less efficiently at his vocation. Finally one attack sticks and he does not improve, notwithstanding the remedies which were formerly effective.

In young men the symptoms which we are apt to label as "neurasthenia," may have been present for a year, two, or more. What was most annoying and could not be relieved by the usual treatment instituted was the languor, the tired feeling which overwhelmed the patient before his day's work was at an end. He may be complaining of cardiac palpitation, indefinite pains in the chest, some cough in the morning, etc. But on the whole he considered himself "run down," and sadly in need of a rest.

In young women the subjective and objective symptoms of chlorosis may have been present for months or years. An examination of the blood has, indeed, shown a low percentage of hemoglobin, and large doses of some iron preparation have been used. Some have had irregularities in the menstrual function, perhaps amenorrhea for several months, and even this was attributed to the anemia. But then they begin to cough; and the cough persists in spite of treatment, when an examination of the chest or of the sputum tells the story.

Others have been "run down" from overwork, physical or mental, for a long time till it is discovered that the cause of their debility is located in the lungs. In many patients the symptoms of dyspepsia are so pronounced as to preclude a careful examination of the chest and they are treated for a long time for "stomach trouble."

This does not exhaust the variety of symptoms which may slowly but surely usher in phthisis. But numerous as they are, they have certain features in common which characterize phthisis in the vast majority of cases, so that if this disease is only borne in mind—and it should, considering its great prevalence—more really incipient cases would be recognized than at present. These clinical phenomena will now be discussed.

**Symptoms.**—Practically all patients with incipient phthisis cough at a very early stage of the disease, and the cases without cough, which have been mentioned by various authors, are rare clinical phenomena, at least they are exceedingly rare among persons under fifty years of age, and may be disregarded. It was already stated that patients who claim that they do not cough are usually individuals who overlook a mild cough, but those around them are apt to notice that they do, and in doubtful cases it is advisable to inquire among those who live with the patient.

A person who never coughed before, but after a "cold" coughs for more than two weeks should excite interest and careful study. If he vomits after fits of coughing, tuberculosis is to be strongly suspected. Paroxysmal coughing spells are also apt to take place during the night and keep the patient awake. Very little expectoration is apt to be brought up at this period—at most some viscid mucus which contains

no tubercle bacilli, nor elastic tissue, though animal inoculation may be effective in disclosing the tuberculous nature of the trouble.

Languor is a constant symptom at a very early stage—the patient feels tired in the morning at rising, but recuperates after working for a few hours. But in the later part of the afternoon he feels fatigued, often drowsy, inclined to sleep. It is this tired feeling which is to be held responsible for the fact that so many patients are erroneously treated for neurasthenia and psychasthenia, or for a “nervous breakdown” for a long time till the true nature of the disease is finally ascertained.

Anorexia is an inconstant and variable symptom of incipient phthisis. In some, especially in youthful subjects, it is very frequent and, coupled with anemia, constipation, etc., is the cause why so many are treated for chlorosis, gastritis, etc. There are many cases in which the appetite is well retained and, when not “dieteted” with a view of improving nutrition and digestion, but urged to eat well and plenty of the foods they are accustomed to eat, they do not lose in weight, but may gain even when the process in the lung goes on actively.

But in the majority of cases a persistent loss of weight is noted at this period. In some it is slow, only one pound per week on the average, while in others it is more rapid and during the first two months fifteen or twenty pounds may be lost.

The activity of the process is best estimated by the fever, which is never absent. It may be slight, only 1° elevation in the afternoon but it can be found in every case by the judicious use of the thermometer. A subnormal temperature during the early morning hours, best looked for by taking it per rectum before the patient leaves his bed, is very frequently observed and of immense diagnostic significance. In many the fever subsides when the patient is kept in bed for a couple of days but reappears as soon as some exercise is allowed. In those with an apparently normal temperature, fever may be provoked by walking or any other form of exercise, as was already discussed in detail (see page 172). In women, the fever may appear only during the menstrual period or a few days before.

In a large number of cases pyrexia is considerable even at this early stage, up to 102° or 103° F. in the afternoon and evening and, measured by comparison with the subnormal temperature in the early morning hours, it is quite high. The “reversed type” of fever with a rise in the morning is occasionally seen.

A significant diagnostic point is that with high fever the patient may not be prostrated as is the case with adults who have fever due to other causes. Moreover, the patient may have a fair, even a good appetite, despite the fact that the thermometer registers 102° or 103° F., which is very rare in fevers due to other causes. During the afternoon access of the fever, the patient, otherwise pale, becomes flushed, often only one cheek is red, he is tired and disinclined to work. But he may keep on working, as was already stated.



Nightsweats make their appearance in a large proportion of cases at this stage. In some they are slight, while in others I have met with profuse nightsweats during the first two weeks of active symptoms. They perspire also at the least exertion or excitement and during a medical examination it is not rare to see large drops of sweat dribbling down the sides of the chest from the axillæ. A constant accompaniment of fever in incipient cases is tachycardia. A case of active phthisis with a pulse rate below 80 per minute is exceedingly rare. In some the heart action is so rapid that they are treated for heart disease or for hyperthyroidism in case the thyroid is enlarged, which is not rare, especially in youthful individuals. While in the early morning after a refreshing sleep, the pulse rate may be normal, the least exertion or excitement will raise it up to 90, 100 or more. Instability of the pulse is characteristic of phthisis. In youthful subjects the tachycardia is apt to be more pronounced than in persons over twenty-five years of age. The blood-pressure is low and a registration less than 70 mm. of mercury is quite common.

Symptoms referable to the respiratory system may not be seen at this stage excepting the cough and at times the intermittent hoarseness, which is usually due to a laryngeal catarrh, or pressure on the laryngeal nerve, and hardly ever to infiltration of the larynx. At times we see patients who suffer from more or less pronounced pains in the chest, especially under the scapula or in the shoulder.

Hemoptysis is quite frequent at this stage. As was already stated, statistics taken of large numbers of patients show that about 10 per cent. of cases begin with hemorrhage. They are the lucky ones, because this clears up the case and proper measures are promptly taken. But many of these initial hemorrhages were actually preceded by a train of symptoms, such as fever, tachycardia, etc., to which the patient paid no attention. However, in about 25 per cent. of cases more or less blood-spitting occurs at the time the disease is recognized. It may be only blood-tinged sputum, a mouthful or two of blood, or even a profuse hemorrhage. It will bear repetition that these hemorrhages are practically never fatal.

**Physical Signs.**—With any or all of these symptoms a diagnosis of incipient tuberculosis should not be made unless physical exploration of the chest discloses a localized lesion in the lungs.

**Inspection.**—Inspection yields excellent diagnostic criteria in most cases at this early stage. Inasmuch as most of the incipient cases are really recrudescences of old quiescent lesions dating back to childhood, we find in many atrophy of the muscles over the site of the lesion. The sternocleidomastoid, the scaleni and trapezius, etc., may be smaller than those on the opposite side and softer, or even flabby to the touch. This is more important to look for than the form of the chest which may be normal, flat, rachitic, etc., without influencing the diagnosis. With the atrophy of the muscles there is usually seen a

slight shoulder-droop and an excavation of the supraclavicular or supraspinous fossa, or at least some flattening and defective motion or lagging of the part of the chest harboring the lesion. This asymmetry, flattening and lagging is very easy to detect if carefully looked for and is, when found, of immense diagnostic importance, provided occupational influences are excluded.

In more recent lesions, or when an old lesion exists in one side but the outbreak of phthisis is due to a new lesion in the opposite side, which is very frequent, we find the muscles over the site of the active new infiltration are tense and rigid, standing out prominently. But this is after all not very frequent, which goes to show that most of the incipient cases are really due to reawakening of old smoldering tuberculous processes in the lung.

**Percussion.**—As was already stated, there are very few cases of active incipient tuberculosis in which no signs of an infiltration can be discovered by careful and gentle percussion. We almost invariably find some airless pulmonary tissue or shrinkage of one apex manifesting itself by a short note or by pulmonary retraction. The height of the apex may be less than that of its mate on the opposite side, or its width may be less, as determined by Krönig's method of percussion. We may also find, though not as often as Krönig believed, that the base on the affected side is somewhat retracted.

In my own experience, percussion signs are more often found over the posterior aspects of the apices than anteriorly. While over the supraclavicular region we may find that the width of the resonant area is less than that of the other side, it is easier and less time-consuming to map out the mesial lines of demarcation between resonance and dullness in the supraspinous fossæ, and over the site of the lesion this line is usually dislocated outward.

It is also easily ascertained whether the height of the apices posteriorly shows any asymmetry. At a very early stage we find that while over the unaffected apex the resonance reaches as far as the interval between the seventh cervical and first thoracic spines, that of the affected apex is much lower. I have found these changes at times before any auscultatory signs made their appearance.

The changes in pitch, duration and intensity of the note obtained at this stage are of less significance than those of shrinkage just spoken of, and they depend too much on the personal equation of the observer to have important clinical bearings. Thus, we often find that a contracted apex is altogether hyperresonant or even tympanitic on percussion, and by comparison the unaffected side appears to emit a defective note. The stories told in text-books about two equally competent clinicians localizing an apical lesion by percussion and each finding it in another side are undoubtedly based on these facts. It is generally due to faulty interpretation of tympany caused by relaxation and hyperfunction of the lung tissue around conglomerations of tubercles, as has already been shown. The discordance may also be due to an

old and cicatrized lesion on one side, while the new and active lesion is in the opposite side of the chest.

Of greater importance is respiratory percussion. The patient is asked to inspire or expire and hold his breath, and we percuss during each phase of respiration. In health the note is clearer during full and held expiration, while over an infiltrated apex a long and held inspiration gives a duller note than found over the opposite unaffected side.

Of the various seats of election of dulness in incipient phthisis which have been described by Lees,<sup>1</sup> Riviere,<sup>2</sup> and others, the sites I have been able to find impaired in most cases at a very early stage are the supra-spinous fossæ near the spine and beneath and above the inner third of the clavicle. Persistent, impaired resonance in any of these places, when accompanied by constitutional symptoms of phthisis, is of great diagnostic significance. Impaired resonance elicited with hooked-finger percussion between the heads of the sternocleidomastoid immediately above the clavicle on one side is very often found.

**Auscultation.**—It is not generally appreciated that the earliest changes in the respiratory sounds in phthisis are modifications of the inspiratory murmur, while changes in the expiratory murmur usually indicate a more or less advanced stage of the disease. At a very early period of the disease the inspiratory murmur loses its soft, breezy character and becomes rough or granular, an indication of partial stenosis of the bronchioles supplying the affected part of the lung or unequal respiratory movement of the infiltrated lung area. In many cases the inspiratory murmur is feeble, at times even absent, over a limited area corresponding to the area of impaired resonance, while the whispered voice is transmitted clearly. But the most common sign of an early lesion is interrupted or cog-wheel breathing, the inspiratory sound is broken up into several parts so that it appears jerky. Either of these types of altered inspiratory murmur may be audible long before the expiratory murmur is in any way changed.

The most common seats of the changed breath sounds are posteriorly near and above the spine of the scapula, the "alarm zone" of Sergent,<sup>3</sup> and rarely in front immediately beneath the inner third of the clavicle. It is located posteriorly as follows: From the centre of the space separating the spinous process of the seventh cervical from that of the first thoracic, a line is drawn as far as the tubercle of the trapezium on the spine of the scapula. From the middle of this line, taken as a centre, a circle is described with a diameter equal to that of a silver dollar. The circumference of this circle forms the boundary of the "zone of alarm" (Fig. 65). When heard at any of these points during ordinary breathing, and repeatedly found on several examinations, not decreasing in intensity but on the contrary

<sup>1</sup> Brit. Med. Jour., 1912, ii, 1268.

<sup>2</sup> Early Diagnosis of Tubercle, London, 1914, p. 25.

<sup>3</sup> Le monde Médical, 1913, xxii, 1121; La Clinique, 1913, viii, 437.

becoming more and more pronounced, rough and cog-wheel breathing are good signs of incipient infiltration of an apex, provided of course, that the constitutional symptoms show activity; otherwise they may be indications of an old and cicatrized lesion. We have already stated that at times feeble breath sounds are found; they may be of a blowing or even of a bronchial character, and some crackling may be audible at the end of inspiration.

Rales are not heard at all over really incipient lesions. Occasionally some sibilation is audible, but this is usually transitory and disappears after the patient takes a deep breath. At most, some dry crackling

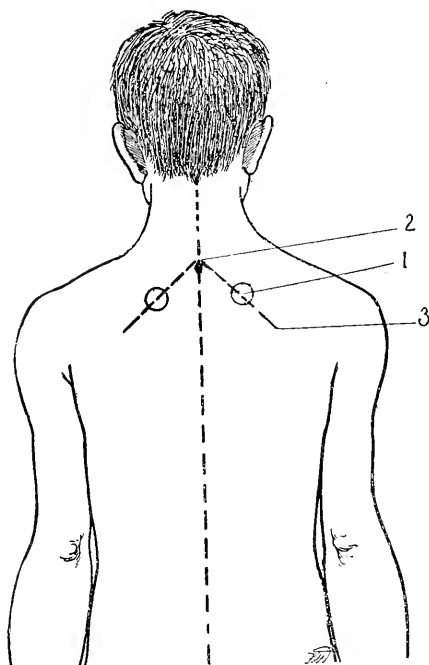


FIG. 65.—1, The "alarm zone;" 2, the space between the spinous processes of the seventh cervical and first dorsal vertebrae; 3, the tubercle of the trapezius.

may be brought out when the patient coughs vigorously. When crepitant and especially moist subcrepitant rales are audible, we are dealing with an extensive lesion of some duration.

In some cases we hear at a very early stage during expiration a hemic murmur originating in the subclavian artery and ascribed to kinking of that vessel by the tuberculous infiltration or by shrinking lung. But it is by no means pathognomonic of phthisis because it is heard in many apparently healthy persons.

The whispered voice is very often transmitted more or less clearly over consolidated areas of lung tissue and when heard when the chest-

piece of the stethoscope is pressed firmly over the skin of the chest, it is of the same diagnostic significance as impaired resonance, with which it usually runs parallel, as has been pointed out by Sewall.<sup>1</sup>

To be of diagnostic significance in early phthisis, the auscultatory signs must be localized over one apex, circumscribed, fixed and persistent for some time, and not influenced by cough and strong respiratory efforts, excepting clicks and rales which may be brought out by cough. Evanescent changes in resonance and breath sounds may be found in many apparently healthy persons. It is for this reason that many who attempt to make a final diagnosis of incipient phthisis during one examination meet with so many failures.

**Elements of Diagnosis of Incipient Phthisis.**—Just as the general and constitutional symptoms, such as cough, fever, tachycardia, emaciation, etc., are insufficient to decide a case till the lesion is localized in the lung, so are the signs obtained by physical exploration of the chest inadequate, even when marked, to prove that we are dealing with a case of active incipient phthisis. Only the combination of both groups of clinical data gives solid support for diagnostic inferences. A skilled diagnostician may easily diagnose a case of advanced phthisis by looking at the pathognomonic facies of the patient, from the history and course of the disease, or from auscultatory findings alone, and only rarely err. But in incipient phthisis it is the correlation of all available clinical data, the history, the symptomatology and course of the disease, combined with the findings of physical exploration of the chest that can be expected to clinch the diagnosis.

The signs enumerated above—defective resonance, narrowing of the resonant areas over one apex, feeble, rough, granular or cog-wheel breathing, or even rales, may each be found in persons of excellent health, at least such as are not actively tuberculous. This is because old and healed lesions, tuberculous and others, leave traces behind them which alter permanently the air content of the pulmonary parenchyma and diagnostic methods in vogue disclose these conditions.

**Sources of Errors.**—I am not prepared to state that the proportion of diagnostic errors made while attempting to recognize phthisis in its very incipiency is greater than in other diseases; in fact, I am convinced that it is not. But in phthisis, owing to its great prevalence and its social aspects, as well as its insidious onset, the opportunities for making mistakes are immense. It is for this reason that the sources of error must be emphasized.

Bias is more often a source of error in phthisis than in any other disease. Especially is this the case when there is a history of exposure to infection. To my mind this is one of the greatest fallacies we have to cope with. It must always be remembered that in large industrial cities everyone is exposed to infection and is, in fact, infected with tubercle bacilli before he passes his fifteenth year. On the other

<sup>1</sup> Jour. Amer. Med. Assn., 1913, lx, 2027.

hand, marital phthisis is less frequent than would be expected if every adult exposed to tuberculosis would become phthisical. Excepting in young children a case must therefore be judged on its clinical manifestations and not on the fact that the patient came into contact with a consumptive.

**Tubercle Bacilli.**—The diagnosis of phthisis is clinched by the finding of tubercle bacilli in the sputum, but is not at all excluded by negative bacteriological findings. Unfortunately, too many wait rather long for the bacilli, thus losing valuable time which often cannot be reclaimed by any known means. Phthisis begins as an infiltration, and only when softening had taken place and the products of tissue disintegration are eliminated through a bronchus, can tubercle bacilli be found in the sputum. Under the circumstances, waiting for tubercle bacilli to make their appearance in the sputum is just as hazardous as waiting for pus to make its appearance through a fistula or sinus before making a diagnosis of a tuberculous joint.

On rare occasions there are errors of quite a different nature. Tubercle bacilli may be found in the sputum of persons who are not actively tuberculous. Of course, from the practical standpoint tubercle bacilli in sputum are an indication that they are in all probabilities derived from a tuberculous lesion in the lower respiratory tract. But in New York City we meet with numerous persons who have reports from some private as well as from the municipal laboratory stating that the sputum of the bearer has been examined and found to contain tubercle bacilli. Yet, without any treatment or special care, they have kept at work for years and felt well. Indeed, many cases are admitted to sanatoriums solely on the strength of positive sputum findings, to be declared non-tuberculous after careful observation.

The reasons for this anomaly are to be sought for in several facts which have not been emphasized as strongly as they deserve. I have no doubt that in busy laboratories mistakes are liable to happen in handling the sputum bottles, in numbering the slides, or while entering the findings in the reports. In banks where the clerks are just as careful as laboratory workers, mistakes occur at times. Even conceding that the number of such mistakes is comparatively negligible, in the individual cases it may count very much.

We have already spoken of the acid-fast rods which simulate tubercle bacilli and which are found in butter and milk, on graminacea, in the soil, in dung and manure, and even in tap water supplied through metal pipes. These bacilli are dead, or non-pathogenic to guinea-pigs, but they give the usual staining reactions. Then we may have the smegma bacilli which have been mistaken for tubercle bacilli and thus have led to erroneous diagnosis and extirpation of healthy kidneys. There are also the acid-fast lepra bacilli, the microorganisms which greatly resemble them and are found in the secretion of the mucous membrane of the nose, also the acid-fast rods found in the saliva and the secretions in cases of bronchitis and pulmonary gangrene. L. Napo-

leon Boston<sup>1</sup> found acid-fast bacilli in patients suffering from acute colds and influenza, and disappearing during convalescence. But most of these microorganisms are difficult to differentiate from tubercle bacilli microscopically, through culture and animal inoculation.

It has recently been found that the spores of lycopodium are acid-fast, so that persons taking pills covered by that substance may impart some of it to the sputum and thus lead to error.

There is a possibility that the acid-fast rods or specks found in the sputum may not have been there before it left the bronchial tubes and trachea, but got into the sputum while it was passing through the pharynx, mouth or lips, especially in persons living in houses inhabited by careless consumptives. It is also important to mention that ordinary smear preparations are less likely to lead to errors of this sort than the antiformin method.

To be sure, the most reliable sign of phthisis is tubercle bacilli in the sputum, and I do not at all intend to underestimate its far-reaching significance. Statistically, the chances of error are undoubtedly insignificant, and a laboratory may be proud that among several thousands of specimens, only half a dozen mistakes have been made. But the practising physician does not treat his patient statistically. In the individual case it is well to bear in mind the possibility of errors of this kind, especially in cases in which the disease does not pursue the course expected in some form of phthisis.

**Skiagraphy.**—Skiagraphy has been discussed in detail in Chapter XVII.

**The Tuberculin Tests.**—The changed reactivity to tuberculin which is observed in organisms infected with tubercle bacilli, which manifests itself mainly by hypersensitiveness to that agent, has been applied in the diagnosis of doubtful cases, especially in sanatoriums. When first introduced it was heralded as specific and it was asserted that finally a positive and uncontrovertible test had been found which decides whether or not an individual is suffering from active tuberculosis.

For diagnostic purposes, tuberculin is applied in various ways. It is introduced directly into the circulation by the subcutaneous method; into the lymph spaces by the cutaneous method, or applied to mucous membranes for normal absorption by the conjunctival method. It has thus been applied to the skin, mucous membrane, and subcutaneously. The subcutaneous application produces general and constitutional symptoms of tuberculin intoxication, while the others produce local effects.

Clinically the following reactions are evoked by the tuberculin test:

1. General reaction, manifesting itself after the subcutaneous injection of tuberculin by fever, chilliness, malaise, headache, backache, etc.

<sup>1</sup> Interstate Medical Journal, 1914, xxi, 330.

2. Focal reaction, consisting in congestive and inflammatory phenomena in the neighborhood of the tuberculous lesion.

3. Local reactions, hyperemia and inflammatory phenomena at the site of the tuberculin application. Of these there are: (a) The cutaneous reaction of von Pirquet and several of its modifications; (b) mucous membrane reactions, such as the ophthalmoreaction of Calmette and Wolff-Eisner, etc., and many others which have been discarded for valid reasons.

**The Cutaneous Tuberculin Test.**—This is the simplest and unquestionably *the* harmless method of application of tuberculin for diagnostic purposes. It is usually performed on the inner surface of the forearm, though any part of the body may do, but it appears that the skin of the trunk is not as sensitive as that of the forearm and thigh. The skin is cleaned with alcohol or ether, and a drop of pure tuberculin is applied. A suitable instrument is then used to make two abrasions, one about two inches away from the spot where the tuberculin has been applied, and the other over the tuberculin. The instrument devised by von Pirquet may be used. It consists of a heavy handle with a spade-like platinum end which is more or less sharp and used for the purpose of scratching or boring a cup-like depression in the skin. It is important that bleeding should not be caused, but only the superficial layer of the skin is scraped away, so as to open the lymph spaces and thus favor absorption of the tuberculin. A needle may be used for the purpose or even the point of a scalpel, making one or two parallel incisions through the superficial layer of the skin. I have found it just as effective to make the abrasion first and then apply the tuberculin with a toothpick, rubbing it vigorously. After five minutes the excess of tuberculin is wiped away with some cotton and the patient allowed to go without any dressing.

If the test turns out negative, it will be seen that twenty-four hours later the two abrasions either heal in the same manner, or when a scab is formed it is of the same appearance on both abrasions. When positive, the control appears healed, or showing a slight scab, while the abrasion to which tuberculin has been applied shows an inflammatory infiltration manifesting itself as a slightly elevated, red papule. This reaction usually appears twelve to twenty-four hours after the application of the tuberculin; on rare occasions it is premature, appearing within four to six hours, and may disappear soon, or remain for days; or it may be late in appearing, even a delay of a week has been observed in rare cases.

The reaction may be slight, showing some redness with infiltration, or a more extensive area of redness with an appreciably raised papule. In some cases the red area is very extensive, simulating erysipelas and the papule is very elevated. Quite often the first test results in a negative outcome, but a second application, about a week later, gives positive results. It is therefore advisable to repeat the test two or three times before pronouncing it unequivocally negative.



PLATE XII



Cutaneous Tuberculin Reaction of v. Pirquet.  
(Taken from Hamill.)



These "secondary" reactions are usually very intense, although the first application was negative. It has also been noted that the tuberculin sensitiveness is often increased by a second or third inoculation and the area at which the first inoculation was made also reacts. Attempts to utilize these facts for diagnostic purposes have not been encouraging.

**Significance of the Cutaneous Tuberculin Reaction.**—Clinical experience has shown conclusively that persons who have at any time been infected with tubercle bacilli react to the cutaneous tuberculin test; experimental investigations have confirmed it. It is immaterial whether the infection is followed by clinical manifestations of disease, or not; whether the tuberculous lesion is active or quiescent, the result is the same. It appears to me, however, that we do not have sufficient evidence for a conclusion as to the question how long after a lesion has healed does the skin remain sensitive to tuberculin. Assuming that no tuberculous lesion ever heals perfectly, which has not yet been proved, we accept that even healed lesions act in this way.

Newborn infants never react to tuberculin, but when living in tubercle-laden surroundings they soon show the hypersensitiveness, as was already shown (page 58). Inasmuch as over 90 per cent. of humanity have been infected before reaching the twentieth year of life, we find that many show positive reactions to tuberculin.

It is thus clear that for clinical purposes, when we look for evidences of active phthisis, this test is of little value, because it shows not only those who suffer from active tuberculosis, but also such as have latent or healed lesions. Moreover, it is negative in rapidly progressing pulmonary tuberculosis, in tuberculous meningitis, in acute miliary tuberculosis and also in the terminal stages of chronic phthisis, when the formation of antibodies is slackened or abolished. It has also been found negative in the presence of other infectious diseases, like measles, scarlet fever, diphtheria, etc., in some cases of pneumonia, and often during pregnancy. In a certain number of cases of undoubted phthisis the cutaneous reaction was found negative without any assignable reason; von Pirquet estimated it at from 2 to 4 per cent., but in my experience it is more than double that proportion.

After many years of experience with this test it was concluded by most authors that a positive cutaneous reaction is of clinical value only in children, and that the younger the child, the more its clinical significance. But from more extensive experience it appears that it is also unreliable in children. From personal experience I am inclined to the conclusion that children between three and fifteen years of age with a positive tuberculin reaction are not necessarily doomed to develop active phthisis; I have even observed many infants under two years of age grow into healthy children in spite of the positive outcome of the test, and the statement of some authors to the effect that an infant under one year showing a positive cutaneous reaction will not survive a year is negated by the many infants I observed

and reported elsewhere,<sup>1</sup> who have thrived despite the fact that during the first six months of their life the reaction was positive.

**Specificity of the Test.**—It appears that from the scientific standpoint the specificity of the test has not been proved to the satisfaction of all, as has already been shown. Autopsy findings by Ganghofner, Radziejewski, Behrend, Bruckner, Reüschel, and many others show that there are cases in which the test was positive, yet no tuberculous lesions were found at the autopsy, and the reverse. Experimentally the evidence is in the same direction (see p. 32).

It has also been found that tuberculin is not the only substance capable of producing a positive skin reaction in tuberculous individuals, but that other toxins when inoculated into the skin often produce changes which are akin to the tuberculin reaction. Rolly<sup>2</sup> found that the skin reacted when inoculated with the toxins of dysentery, typhoid, paratyphoid, pyocyanous, cholera, etc. Just as with tuberculin, these toxins were always negative in very young infants, and in children suffering from acute infectious diseases, as scarlet fever, measles, etc., becoming positive during convalescence. The controls, performed with carbol-glycerin, were always negative. In short, these non-tuberculous toxins showed all the characteristics of tuberculin when inoculated into the human skin. That any or all of these toxins acted in an anaphylactic or specific manner may be ruled out because, with the exception of tuberculosis, the individuals tested never suffered from typhoid, paratyphoid, cholera, diphtheria or pyocyanous sepsis. Tenzer<sup>3</sup> obtained skin reactions indistinguishable from those of the von Pirquet test with cholera vaccine and with a mixture of peptalbumoses, in persons in whom the tuberculin test was positive.

From these experiments, as well as from those performed by Sorgo,<sup>4</sup> it appears that tuberculous individuals react with a specific intensity to tuberculin and to other toxins, thus indicating that it is mainly due to hypersensitiveness of the skin. The assumption that the skin of the tuberculous is endowed with a specific allergy to tuberculin alone is thereby disproved. The allergy is evidently a cutaneous hypersensitiveness to the action of toxins in general. Hamburger,<sup>5</sup> one of the most authoritative champions of the specificity of the tuberculin test, after inoculating tuberculous patients with substances similar to those with which tuberculin is prepared (glycerin, bouillon, extractives, salts, etc.) became convinced that the cutaneous reaction is due more to the latter substances than to the tuberculin which acts merely as a skin irritant.

We are therefore justified in concluding that we are far from having sufficient and satisfactory information to speak with certainty

<sup>1</sup> See *A Study of the Child in the Tuberculous Milieu*, *Arch. of Pediatrics*, 1914, xxxi, 96, 197; 1915, xxxii, 20.

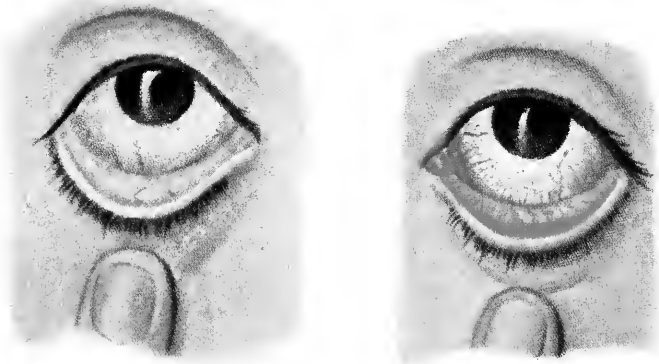
<sup>2</sup> *Münch. med. Woch.*, 1911, lviii, 1285.

<sup>3</sup> *Monatsschr. f. Kinderheilkunde*, 1911, x, 131.

<sup>4</sup> *Deut. med. Wchnschr.*, 1911, xxxvii, 1015.

<sup>5</sup> *Die Tuberkulose des Kindesalter*, p. 37.

PLATE XIII



Tuberculin Ophthalmo-reaction. (Taken from Citron.)



about the cutaneous tuberculin test and its underlying causes, and from the theoretical standpoint its specificity has not been proved conclusively.

However, clinically the test is important in showing the wide dissemination of tuberculous infection among civilized humanity, even though the same results could be also obtained with substances other than tuberculin. In children it shows whether they have been infected with tuberculosis and in infants it even points to active tuberculosis, but in adults it is of no clinical value at all.

The various modifications of the cutaneous tuberculin tests are not superior to the von Pirquet method. The Moro test, consisting in rubbing tuberculin ointment into the skin is of less value than the one described above. The percutaneous, the quantitative cutaneous test, etc., offer no advantages over the von Pirquet test, which is after all the simplest and most reliable.

**The Conjunctival Reaction.**—The conjunctival reaction invented by Calmette and Wolff-Eisner, is made by instilling into the conjunctiva, with an ordinary eye dropper, one drop of a 1 per cent. solution of tuberculin. The reaction appears within twelve hours and reaches its optimum in twenty-four hours, producing redness of the palpebra, and when the reaction is intense, the redness is more pronounced and there is also injection of the vessels of the eyeball and more or less well-marked secretion of mucus. It may last for two or three days. Of course, in estimating the effects of the tuberculin, comparison is made with the other eye.

Among clinically non-tuberculous persons, from 10 to 25 per cent. react, while among those who are evidently tuberculous, between 50 and 75 per cent. show a reaction with this test. It has been practically discarded of late because in many cases inflammatory phenomena have appeared in the tested eye which are quite troublesome. In one of my cases the inflammation was so severe, persisting for three months, that I have ever since hesitated in applying it. Bandelier and Röpke state that experiments on animals have shown that this test is unreliable in cases of human phthisis, since the reaction may be negative in spite of the presence of active tuberculosis unless 10 per cent. solution of tuberculin is used, and this should not be done when dealing with human eyes.

**The Subcutaneous Tuberculin Test.**—This is the test preferred by most of those who have confidence in the diagnostic value of tuberculin in doubtful cases. It is claimed that it is not only reliable in deciding whether the patient has ever been infected with tubercle bacilli, but also in showing whether the disease is active and that in many cases it even shows the area involved at the time the test is made by the so-called "focal reaction."

Of the various ways in which it is performed, the following is the simplest and gives the same results as any that has been devised:

For twenty-four hours the temperature of the patient is taken every

three hours and carefully recorded. Inquiries are made as to the subjective symptoms, especially pains in the chest, headache, cough, expectoration, etc. An injection of 0.1 mg. of tuberculin is then made subcutaneously in the region of the back below the angle of the scapula, or any other place. Of course, all antiseptic precautions are to be rigidly observed and the skin washed with alcohol or ether. In case no reaction appears within forty-eight hours, a second injection is made with the same amount of tuberculin, while some increase it to 1 mg. This dose is again increased in case no reaction follows to 5 mg. and even to 10 mg. in case the test proves negative and a fourth injection is given. Of course, in children smaller doses are used.

**The Reaction.**—Usually between ten to twelve hours, rarely between six to eight hours, in case the reaction is positive, constitutional, local, and focal symptoms make their appearance. Some say that it may be delayed as long as forty-eight to seventy-two hours, but this must be very rare; I have never encountered it. Of the constitutional symptoms, fever is the most constant and reliable. The temperature begins to rise six to twelve hours after the injection, reaching 100° to 102° F., and in those showing a severe reaction, it may even go up to 104° F., and I have seen several cases in which it was higher. There are usually constitutional symptoms of hyperthermia—headache, backache, pains in the joints, weakness, malaise and, in some cases, nausea and vomiting. Rarely the prostration is very pronounced, while in others it may be slight, or even absent, irrespective of the degree of fever. These symptoms usually subside within twenty-four to forty-eight hours and only rarely last longer.

At the site of the injection the local reaction manifests itself in tenderness or even pain, redness, and swelling, which may be small—only about 1 cm.—but in some cases the infiltration is as large as a hen's egg. Lymphangitis and enlargement of the regional lymphatic glands may occur.

The so-called "focal reaction" is very rarely observed in phthisis. It is said to consist in congestion of the lesion in the lung, an increase in number and consonance of the rales, a change in the breath sounds, and extension of the dull areas, accompanied by an increase in the cough and expectoration. Tubercle bacilli hitherto absent from the sputum may now be found. My own experience leads me to the conviction that this focal reaction is very unreliable. It occurs but rarely, and when we recall that in phthisis the physical signs change so often, and that a skilful clinician one day finds signs in one side and the next day in another without tuberculin injections, we may always suspect that the focal reaction is not necessarily a result of the tuberculin injection, at least its inconstancy should lead us to this conclusion.

**Clinical Value of the Test.**—The object of the test is to clear up doubtful cases in which there are symptoms and signs pointing to active phthisis but which are not convincing to clinch the diagnosis. In such cases, the advocates of the test claim that a positive reaction decides



in favor of active disease, while a negative outcome positively excludes it. It has been used mostly in sanatoriums for these purposes.

Careful analysis of the conditions under which this test is negative or positive shows that it is hardly of greater reliability than the cutaneous or conjunctival test. Investigations by Franz,<sup>1</sup> Hamman and Wolmann,<sup>2</sup> Beck,<sup>3</sup> and many others show that it may be positive in healthy persons who do not develop phthisis subsequently. The experience of all who have applied this test to large numbers of actually or apparently non-tuberculous individuals is the same as that of Franz, Hamman and Wolman, Beck, etc. It is always found that between 40 and 60 per cent. of humanity react to the subcutaneous tuberculin test, providing it is repeated with ascending doses three or four times.

**Specificity of the Test.**—We have already mentioned that many non-tuberculous substances have a toxic action on the organism infected with tubercle bacilli. Thus, according to experiments by Mettetal,<sup>4</sup> Preisich and Heim,<sup>5</sup> Petruschky,<sup>6</sup> and many others, nucleins, blood-serum, testicular extract from healthy animals, culture-free bouillon, and other foreign albumoses, when injected into tuberculous persons, may provoke reactions not unlike the general reaction of tuberculin. It appears that the tuberculin reaction is part and parcel of the hypersensitiveness of the infected organism to foreign proteins of any kind, tuberculous and non-tuberculous (see p. 33).

**Diagnostic Value.**—Considering that the subcutaneous tuberculin test discloses latent infection as well as active tuberculous disease, its diagnostic value is limited, bearing in mind that over 90 per cent. of humanity have been infected at some period of their life. What we look for is active disease and when the test also shows those who are not phthisical, its value in diagnosis is limited indeed.

“A positive tuberculin reaction,” say Hamman and Wolman, “is merely confirmatory evidence and never decides with certainty an otherwise doubtful diagnosis. Indeed we feel that caution is decidedly in place not to lay too much emphasis upon a positive reaction, for if a patient is suffering from symptoms which may be accounted for by a number of different conditions, and by applying the test we admit such uncertainty, a positive reaction does not impel the conclusion that these symptoms are due to tuberculosis. If such a large percentage of healthy individuals harbor clinically unimportant tuberculous lesions, a certain proportion of those suspected of having tuberculosis must likewise harbor them, though the symptoms that attract our attention may be due to some other disease.” With this view the present writer agrees entirely.

<sup>1</sup> Wien. klin. Wchnschr., 1909, xxii, 991.

<sup>2</sup> Tuberculin in Diagnosis and Treatment, New York, 1912.

<sup>3</sup> Deut. med. Wchnschr., 1899, xxv, 137.

<sup>4</sup> Valeur de la tuberculine dans le diagnostic de la tuberculose de la premiere enfance, Thèse de Paris, 1900.

<sup>5</sup> Zentrablatt f. Bakteriologie, 1902, xxxi, 712.

<sup>6</sup> Ergebn. d. Inn. Med. u. Kinderheilk., 1912, ix, 557.

**Dangers of the Test and Contra-indications.**—The subcutaneous tuberculin test is not without dangers. When carelessly performed with excessive doses, latent or quiescent lesions may be flared up into activity. Recently, L. Rabinowitsch,<sup>1</sup> Bacmeister,<sup>2</sup> Leo Kessel,<sup>3</sup> and others have shown that living and virulent tubercle bacilli may appear in the blood after an injection of tuberculin. In some cases it has been observed that hemoptysis is provoked by the test, and all agree that it must not be given during or soon after a pulmonary hemorrhage. In general the reaction consists essentially in a transient toxic injury to the body, and the nervous system bears the brunt of the traumatism.

It has also been found dangerous in cases of heart disease, arteriosclerosis, nephritis, diabetes, etc. In epileptics it has been observed that the reaction may provoke convulsions. Even Bandelier and Röpke say that it is contra-indicated when miliary tuberculosis is suspected "since its downward course might be accelerated." Sahli,<sup>4</sup> who uses tuberculin for therapeutic purposes extensively, says: "The use of tuberculin for diagnostic purposes ought to be condemned. It is unreliable both positively and negatively. Diagnostic injections are dangerous."

**The Complement-fixation Test.**—Quite recently the complement-fixation test on the lines of the well-known Wassermann reaction for syphilis has been applied in the diagnosis of tuberculosis. It has been studied by Besredka and Manoukhine,<sup>5</sup> Calmette and Massol,<sup>6</sup> Debains and Jupille, in France, and in England by James McIntosh, Paul Fildes,<sup>8</sup> J. A. D. Radcliffe and Edward Glover.<sup>9</sup> In this country, J. Bronfenbrenner,<sup>10</sup> A. M. Stimson,<sup>11</sup> Charles F. Craig,<sup>12</sup> and others have reported good results with this test.

But so far the results appear to be conflicting in certain points, so that further careful research, combined with clinical observations are necessary before deciding on the specificity and clinical applicability of the test in general practice. The main difficulty is evidently the fact that different authors have used different antigens. Besredka used one prepared from egg-broth cultures of tubercle bacilli; Radcliffe used a freshly prepared unsterilized emulsion in saline solution of living tubercle bacilli grown on glycerin-egg medium; Hammer used an alcoholic extract of tuberculous tissue to which was added a certain amount of old tuberculin; Stimson and Bronfenbrenner use

<sup>1</sup> Berl. klin. Wehnschr., 1913, l.

<sup>2</sup> Münch. med. Wehnschr., 1913, lx.

<sup>3</sup> Amer. Jour. Med. Sci., 1915, cl, 337.

<sup>4</sup> Fifth Confer. Nat. Assn. Prev. Consumption, London, 1913, p. 57.

<sup>5</sup> Ann. de l'Inst. Pasteur, 1914, xxviii, 569; Compt. rend. Soc. de Biologie, 1914, lxxvi, 197.

<sup>6</sup> Ann. de l'Inst. Pasteur, 1914, xxviii, 338.

<sup>7</sup> Compt. rend. Soc. de Biol., 1914, lxxvi, 199.

<sup>8</sup> Lancet, 1914, ii, 485.

<sup>9</sup> Quarterly Jour. of Medicine, 1915, viii, 339.

<sup>10</sup> Arch. Intern. Med., 1914, xiv, 786; Proc. Soc. Exper. Biol. and Med., 1914, xii, 48.

<sup>11</sup> Bull. 101, Hyg. Laborat., U. S. P. H. S., 1915.

<sup>12</sup> Amer. Jour. Med. Sci., 1915, cl, 781.

Besredka's antigen; Craig's antigen consists in an extract of several strains of human tubercle bacilli prepared by a special method. It is thus clear that with so many different methods, the results are hardly comparable. Moreover, as McIntosh points out, Besredka's antigen cannot be considered absolutely specific since Inman and Küss, and Leredde and Rubinstein, found that non-tuberculous syphilitics gave the reaction frequently. Even if the explanation that it is due to the lipoids derived from the egg constituents of the medium which react with the syphilitic serum in a manner similar to tissue-extract antigen, is correct, it does not help us in our efforts to find a specific test for active tuberculosis.

Various authors report between 40 and 95 per cent. of positive results with the complement-fixation test. Some state that a positive reaction means an active tuberculous process somewhere in the body. McIntosh and Fildes state even that a small lesion may not reveal itself by this test; "the lesion must be of considerable dimensions before the reaction can detect it. A caseous bronchial gland will not give a positive reaction; indeed, the common affection of the cervical glands will usually yield a negative result. On the whole, we have come to the conclusion that a lesion in order to give positive results must be of such dimensions as to constitute 'disease' and require the intervention of the physician or surgeon. We look upon the positive reaction, therefore, as indicating 'active tuberculosis.'" On the other hand, Craig found that 65 per cent. of clinically inactive cases of pulmonary tuberculosis gave positive reactions. Most writers obtained positive reactions in patients with syphilis.

The test deserves further trial and even if it proves that it has the limitations of the Wassermann reaction it will be invaluable. Meanwhile some important points are to be borne in mind while interpreting the findings. Glover shows that with a healed lesion "positive fixation may occur even for some years afterward when the history and symptomatology do not point to a progressive lesion." In other words, a diagnosis of active phthisis should not be made in the absence of symptoms pointing to disease, though this test is positive. McIntosh and Fildes also state that "a positive result indicates tuberculosis with certain group exceptions." The fact that nearly all syphilitics without active tuberculosis give positive fixation reactions, has already been mentioned. Another fact is to be borne in mind: Antituberculin is not constantly present in the sera of tuberculous individuals, and for this reason the reaction is at times negative in patients with active tuberculosis. Indeed, it appears that in the later stages of phthisis it is, as a rule, negative; experimental evidence is in agreement with these clinical observations.

**Other Special Tests.**—Most of the other special diagnostic tests which have been brought forward from time to time have been found wanting in reliability; their limitations preclude their general adoption. Arneth's blood-picture has never been considered of diagnostic

value and was only urged as of prognostic significance (see p. 225). Wright's opsonic-index method has been given a very extensive trial, especially in English-speaking countries, but has been found unreliable. The results are very conflicting and the method is altogether unsuitable for general adoption.

### DIFFERENTIAL DIAGNOSIS.

Incipient phthisical lesions are to be differentiated from three groups of non-tuberculous apical lesions:

1. Collapse induration.

2. Apical catarrhs, often manifesting themselves after attacks of influenza, or found in persons suffering from pulmonary emphysema, or who are of defective muscular development, especially women and those who work at indoor or dusty trades.

3. Apical indurations found in persons with heart disease.

**Collapse Induration of the Apex.**—The two apices are not always symmetrical, nor do they always have the same resonance and breath sounds in apparently healthy people. In some, the differences are so striking as to attract attention, and when fever, cough, etc., occur for any reason, a diagnosis of phthisis is apt to be made, based upon these asymmetrical findings over the upper parts of the chest. In addition, there is quite often met with, collapse of the apical parenchyma, resorption of the air with subsequent induration, which greatly simulates a phthisical lesion. Krönig<sup>1</sup> first described these cases in detail. It is a purely local, non-infectious lesion, showing impaired resonance or even dulness, rough inspiratory murmur, harsh or prolonged expiration and even true bronchial breathing with some dry crackling over one apex, usually the right. These physical signs are at times enough to mislead into a diagnosis of tuberculosis, especially if the patient states that he had some blood-streaked sputum, which may happen during an acute cold.

The points of differentiation between collapse induration and phthisis are the following: Patients showing signs of the former have been sufferers from nasal obstruction since childhood and generally have enlarged turbinated bones, nasal polypi, adenoids, or enlarged tonsils. They complain that they have not been able to breathe properly through the nose for years, have expectorated considerably, suffered from dryness and itching of the throat, and have had a strong tendency to colds, tonsillitis and frequent bronchial catarrh. The classical face of the mouth-breather is often seen in these patients—open mouth, enlarged and drooping lips, absence of the nasolabial fold, etc. In tuberculosis all these are usually absent. In addition to the absence of tubercle bacilli, the sputum shows distinct evidences that it is derived from the upper respiratory tract: It is watery, mixed with saliva and

<sup>1</sup> Deutsche Klinik, 1907, xi, 634.

colorless; sometimes containing gray or bluish globules, not unlike the kind seen in pneumokoniosis. Microscopically there are often found epithelial cells from the mouth, nose and throat, but no tubercle bacilli.

The physical signs may exquisitely simulate those of phthisis. The suggestion of Krönig that in phthisis the base of the affected lung is always more or less adherent has not been verified by the writer. The general appearance of the patient is rather good. In spite of the fact that he has been coughing for months or years, he is well nourished and does not lose weight, as is usually the case in active phthisis. He is able to keep at work, and the sense of fatigue and languor characteristic of tuberculosis is lacking. Moreover, there is no fever, which can be discovered in every case of active phthisis. Nor is there tachycardia and instability of the pulse.

**Apical Catarrh.**—Most of us have been warned against the term apical catarrh of a non-tuberculous nature as something which does not exist and should be banished from medical terminology. But it appears that during recent years the profession is again acknowledging that there is often to be seen a catarrhal condition of one or both apices which is not caused by tubercle bacilli. Among workers at dusty trades it is quite frequent, especially those who have pulmonary emphysema. After attacks of influenza there is very often left a persisting catarrh of one or both apices. In persons having emphysematous lungs, dulness of the apices, especially the right, due to local bronchitis and tracheitis, is very common. The difficulty of differentiating these cases from phthisis is evident when we mention that in our hospital work we quite often find them admitted as advanced consumptives, and only after observation extending over several weeks are we ready to discharge them as non-tuberculous.

They are distinguished from phthisis by the absence of tubercle bacilli from the sputum, by the normal temperature and pulse, and the blood-pressure which is often elevated in emphysematous persons over forty years of age, while in tuberculosis it is low. In young persons with weak muscles, when signs of apical catarrh are encountered the diagnosis is often difficult and requires prolonged observation. The absence of the constitutional symptoms of phthisis points to their non-tuberculous character.

**Pneumonic Processes.**—The most baffling cases which simulate phthisis to a degree as to prove perplexing at times are those which are caused by pulmonary infection with various cocci. In some only observation for weeks will clear up the case. In this class belong the localized catarrhs of the apices remaining after attacks of influenza, while in others they originate primarily after some exposure, etc. D. Finkler<sup>1</sup> was the first to make a careful study of these streptococcus infections of the lung; Albert Fraenkel<sup>2</sup> also describes them, and in

<sup>1</sup> Infektion der Lunge durch Streptokokken und Influenzabazillen, Bonn, 1895.

<sup>2</sup> Spezielle Pathologie und Therapie der Lungekrankheiten, Berlin, 1904, p. 798.

this country they have been described by David Riesman,<sup>1</sup> William Charles White,<sup>2</sup> and the present writer.<sup>3</sup> The patients cough, expectorate more or less sputum, have fever; at times the temperature curve is not unlike that common in incipient phthisis, and there may even be nightsweats, anorexia, and loss in weight. These symptoms may keep on for several weeks or even two or three months. In many cases the process is localized in one of the lower lobes of the lungs and manifests itself by impaired resonance, bronchovesicular, and at times pure bronchial breathing over a limited area, and rales, which may be moist and consonating. After a protracted illness the patient invariably recovers and all the local signs disappear.

In the cases in which the lesion is in a lower lobe, the diagnosis should not be difficult. It must never be lost sight of that initial tuberculous lesions are almost invariably localized in the upper lobes of the lungs, and it requires great courage on the part of a physician to diagnose tuberculosis with an initial lesion in a lower lobe, unless there is positive sputum. This is a point which cannot be emphasized too strongly. In these pneumonic processes, the sputum lacks both tubercle bacilli and elastic fibers.

Greater difficulties are presented when these pneumonic processes are localized in one of the pulmonary apices; at times only prolonged observation clears up the case. Such cases have been reported by Külbs,<sup>4</sup> Friederich Müller, White, and others. I see them very often in New York City treated at the tuberculosis clinics, and have known of some who have been banished to sanatoriums or distant climates. They usually occur in persons who have been emphysematous, especially such as have been mouth-breathers, or have had bronchitis, or tracheitis for many years. The only criteria for diagnosis which have helped me are the following: The sputum is negative as regards tubercle bacilli and elastic tissue; the fever which is observed the first week or two after the onset of the cough disappears soon, and nightsweats are very rare. The appetite of the patient is usually good and, inasmuch as he is frightened by being classed as a "suspect," he soon begins to take care of himself and eats well, so that he gains in weight. But what is of most importance is that the rapid pulse of incipient phthisis is lacking. We have already stated that tuberculosis without tachycardia, or at least instability of the pulse, is exceedingly rare. In short, a diagnosis can be made by careful clinical observation of the constitutional symptoms for a few weeks.

William Charles White recently reported a case of diphtheria of the lung which exquisitely simulated incipient phthisis.

**Apical Induration with Cardiac Disease.**—Persons suffering from heart disease, especially mitral stenosis, often cough, spit blood, have mild

<sup>1</sup> Amer. Jour. Med. Sci., 1913, cxlvi, 313.

<sup>2</sup> Trans. Nat. Assn. Study and Prev. Tuberc., 1915, xi, 140.

<sup>3</sup> Nontuberculous Apical Lesions, New York Med. Jour., 1913, xcvi, 13.

<sup>4</sup> Ztschr. f. klin. Medizin., 1912, lxxiii, 169.

fever and emaciation, and are often treated for tuberculosis. We have already mentioned that next to tuberculosis, mitral stenosis is a frequent cause of hemoptysis (see p. 196). When the lungs are carefully examined, it may be found that, because of the degeneration of the cardiac muscle, there is produced a passive congestion or even induration of one or both apices. In some the clinical picture simulates that of phthisis to a remarkable degree.

Mistakes of this character can be avoided by carefully examining the heart in every case and, while it is possible that patients with mitral stenosis should become tuberculous, yet this is very rare. In fact, it has been my rule never to diagnosticate tuberculosis in one showing disease of the mitral valve unless the sputum reveals tubercle bacilli, or finding signs of pulmonary excavation on physical examination. Mitral disease is almost always accompanied by cardiac hypertrophy, which is never seen in phthisis.

**Chronic Bronchitis and Bronchiectasis.**—In these conditions the changes in breath sounds and the rales are distributed all over the chest, especially the lower parts, and are only rarely localized at the upper parts of the thorax, when they may be mistaken for phthisis. The abundant sputum separates into three layers, contains no tubercle bacilli, and the general condition of the patient is rather good, while with tuberculosis of such extensive distribution, there would be fever, emaciation, etc. Bronchiectasis is occasionally very difficult to differentiate from phthisis when hemoptysis occurs. In many cases we must wait till the hemorrhage ceases for a careful examination which shows that in bronchiectasis the lesion is located, as a rule, in the lower parts of the lungs and not in the apex; the sputum contains no tubercle bacilli nor elastic tissue, which is never absent in phthisis, and the history of the case shows that the patient has coughed and expectorated for many years, perhaps since childhood, yet his general condition is fair or even good. On the other hand, in phthisis the active symptoms of disease begin later in life, and with extensive lesions there is emaciation, debility, and especially tachycardia.

More difficult is it to diagnosticate phthisis implanted in a chest with chronic bronchitis or pulmonary emphysema. In some cases only the clinical course—the emaciation, the fever, night sweats, etc.—and particularly the bacteriological findings decide.

**Syphilis of the Lung.**—This often exquisitely simulates phthisis. It is, however, a very rare disease. Osler found only 12 cases of syphilis of the lung in 2800 postmortems at the Johns Hopkins Hospital. In 8 of these the lesions were in congenital syphilis. In 11 cases there were definite gummata. Clinically the presence of syphilis of the lung was suspected in only 3 of the cases. When it occurs, it may be distinguished from phthisis by the fact that in the former the lesion is usually at the base, the Wassermann reaction and the absence of tubercle bacilli from the sputum. In some the therapeutic test alone decides.

These two diseases occur very often together, and it appears that syphilis modifies the course of phthisis rather favorably, probably because it is characterized by the tendency to the formation of connective tissue. It should always be remembered that the presence of syphilis does not exclude phthisis, but that the latter is very often engrafted on the former.

**Duration of the Incipient Stage.**—Incipient phthisis is also called “early” phthisis, and thus a confusion is caused in the minds of the laity, as well as of physicians who assume that a case is incipient only for a certain time and then progresses to the second or third stage, unless properly treated. This is wrong. There are cases which are “advanced” soon after the active symptoms manifest themselves, while others, though remaining active for years, never pass beyond the stage of incipiency. Indeed, we meet with many patients who have been tuberculous for many years, and have been admitted to sanatoriums several times as “early” cases.

The sagacious clinician, Laennec, stated nearly one hundred years ago that it appears to him that hardly any consumptive succumbs to the first attack of the disease, and that in the vast majority of cases the first attack is erroneously diagnosed as a mild respiratory trouble. The disease then remains latent for a longer or shorter time to break out again, perhaps with greater severity. Many years of research along scientific lines have confirmed Laennec’s observation. A large number of cases never become “advanced” in the sense we use this term. Others show greater activity, and the process in the lungs proceeds from infiltration to caseation, softening and excavation within six months or a year. A larger proportion of active cases remain quiescent for one or two years, and then suddenly take a turn for the worse and the patient sinks, succumbing to exhaustion or to some complication.

On the whole it may be stated that “incipiency” does not necessarily imply earliness of the process. It means a limited and circumscribed lesion which is not manifesting a tendency to acute progression, but either remains quiescent or leans to cicatrization of the lesion. In this stage the patient may remain for many years, and no average duration can be assigned. It can only be estimated in the individual patient, depending as it does on so many different and complex factors which have been discussed elsewhere in this book.



## CHAPTER XX.

### CHRONIC PHTHISIS. ADVANCED STAGES.

**Course of Incipient Phthisis.**—In a large proportion of cases phthisis does not pass beyond the stage of incipency. The patient coughs, expectorates, has fever, hemoptysis, etc., for several weeks or months, and, after taking a rest in the country, spending a few months in a sanatorium, or even while continuing at his occupation, he slowly recuperates and recovers, never to be troubled again with pulmonary symptoms. In most of these cases there are left remnants of the pulmonary lesion in an apex, manifesting themselves in the shape of impaired resonance, some prolonged expiration and sibilation. This conforms to the abortive type of tuberculosis which will be discussed later on (Chapter XXIII).

But in many cases the disease progresses steadily, especially when no proper treatment has been instituted, and occasionally irrespective of the treatment. In a small proportion of cases the progress is rather rapid and within one or two months after the first symptoms have appeared the patient is a confirmed consumptive; while in others the course is slower, the patient keeps on coughing, expectorating, losing flesh and strength for several months or years, when a change takes place and he is apparently improved or cured, or he succumbs to the disease.

In the vast majority the progress of the disease is marked by distinct remissions, during which the patient feels comparatively well, is able to pursue his vocation, and he, as well as his physician, are under the impression that a permanent cure has been attained, to be undeceived now and then by the appearance of an acute exacerbation of the disease during which the patient is laid up for several days or weeks, or by a pulmonary hemorrhage which may or may not be copious; an attack of pleurisy, with or without effusion, etc.

There is another class of cases in which the focus in the lung remains quiescent, but does not cicatrize for many years. Physical examination of the chest shows distinct signs of an active pulmonary lesion and an examination of the sputum may even disclose tubercle bacilli, but the symptomatology and course are benign—the cough is mild, there is no fever, no night sweats, no emaciation, and the patient is capable of working at his vocation for years.

**Oscillating Course of Chronic Phthisis.**—A continuous course from bad to worse till the patient dies, or with improvement till he recovers, is uncommon in chronic phthisis. It is characteristic of

either the abortive form of phthisis, on the one hand, or of acute galloping phthisis, on the other. But the usual case of chronic phthisis pursues a discontinuous, paroxysmal, I may say a capricious course, marked by periods of acute or subacute exacerbations of the symptoms, and periods of remissions during which the patient is more or less free from the troublesome symptoms, or he may even feel comparatively well, working efficiently, especially if he is engaged in some intellectual pursuit. I have seen many who have worked at hard manual labor for months until an acute exacerbation laid them up for several weeks, but they sooner or later recuperated and went to work again until another acute exacerbation interfered.

These acute exacerbations during the course of chronic phthisis usually have morbid anatomical substrata. In active phthisis the affected part of the lung caseates, softens and is finally eliminated by cough and expectoration, leaving a fistula to drain the excavation, surrounded by a fibrous capsule which inhibits or prevents absorption of toxic matter. The patient may feel comparatively well as long as the cavity in the lung is well drained. But now and then the fistula is obstructed, or a new area becomes involved by contiguity or metastasis, and again acute symptoms of constitutional toxemia make their appearance. This acute exacerbation keeps on for some time till either the fistula opens again, or the newly involved area has softened and the products of tissue disintegration are eliminated and the patient feels well again, though he is by no means cured.

This undulating course of phthisis can be clearly observed by studying the temperature, expectoration, emaciation, etc., of the patient, as was done by Bezançon,<sup>1</sup> Serbonnes<sup>2</sup> and others. It may keep on for many years. In most cases one of two things finally occurs—either the infiltrated or excavated area in the lung cicatrizes or becomes encapsulated and shrinks and the disease is arrested; or during one of these exacerbations the pulmonary involvement becomes too extensive and can no more become quiescent and, with or without some complication, the patient succumbs.

We may say that during the long course of chronic phthisis there is an intense struggle between the bacilli and the resistance of the host. We have seen that everybody possesses more or less resistance; else every infection would speedily prove fatal. In this struggle the bacilli gain the upper hand for a time and cause an acute exacerbation, but the innate resistance is again called upon and usually responds, the result being a truce, until the bacilli again catch the organism napping. The final outcome depends on many and complex factors which are discussed elsewhere.

**Symptoms.**—The cough, which may have been mild during the incipient stage, gradually becomes more and more annoying and productive. It may be painful, paroxysmal and exhausting, and end

<sup>1</sup> Paris Médical, 1911, p. 133.

<sup>2</sup> Les Poussees évolutives de la tuberculose pulmonaire chronique, Paris, 1910.

in vomiting, especially after the evening meal. But with the advance of the process the cough is ameliorated in most cases; while it does not cease altogether, it becomes "looser;" the sputum is brought up without great effort. During acute exacerbations it is usually aggravated, often painful due to complicating pleurisy, etc. In some cases the cough is mild throughout the course of the disease, while in others it constitutes the main complaint of the patient. In fatal cases it may be absent during the last few days of life, when the reflexes are abolished, or because of severe emaciation and muscular atrophy, the patient has not enough strength for the efforts at coughing.

The mucoid sputum of the incipient stage becomes more and more mucopurulent with the advance of the disease and almost invariably contains tubercle bacilli. Exceptionally, none are found in a case that keeps on progressing, even to fatal issue. Elastic fibers are, however, found in practically all cases in which the disease has passed incipiency, owing to the destruction of lung tissue during caseation and liquefaction. Immediately before and during an acute exacerbation the amount of sputum may be diminished, but within a few days it again increases in quantity. With the disintegration of lung tissue and formation of vomicae, the character of the sputum changes; it becomes thick, nummular and sinks in the water of the receiving vessel. During hemorrhages it is sanguineous, and often without any evident hemorrhage it is tinged with blood. During quiescent periods the amount expectorated is, as a rule, diminished; it may lose its purulent character and, when a cure is established, the expectoration may cease. In fatal cases we often note that during the last few days little sputum is brought up. The patient has not sufficient strength to expel it.

The temperature in active advanced cases is not of a characteristic type. In progressive cases it may be continuous or remittent till the end—recovery or death. Usually the curve, when studied for several months continuously, pursues an undulating or cyclic course. For several weeks it is high, no matter what type it is, rising to 101° or even 104° F. in the afternoon, and declining several degrees in the morning, in many cases even to a subnormal degree. Slowly an improvement is noted, the temperature becomes lower and lower and we may find a period of either subfebrile or even normal temperature for a few weeks. In many cases I have noted a subnormal temperature for comparatively long periods.

But suddenly—perhaps after a chill or some indiscretion—or gradually, the temperature rises again and keeps at a high level for several days or weeks, thus marking an extension of the process to a hitherto unaffected area of the lung, or some complication.

It is noteworthy that during the afebrile periods the patient feels quite well and for weeks may consider himself cured, to be sadly disappointed during the acute exacerbations which are sure to come in most cases. Even during febrile periods many feel comparatively

well and have a good or fair appetite as was already stated. The intellect is usually clear; those engaged in intellectual pursuits may follow their vocations during the exacerbations. I have had patients who did business on a high scale under such circumstances, and writers and artists who produced their best work while the thermometer registered 103° F. The euphoria, which is characteristic of phthisis, is best observed in far-advanced cases.

Emaciation goes hand-in-hand with other constitutional symptoms, especially fever. Those who have no quiescent periods lose flesh very rapidly and within a few months may be reduced to mere skeletons. In those in whom the disease runs an undulating course, we often

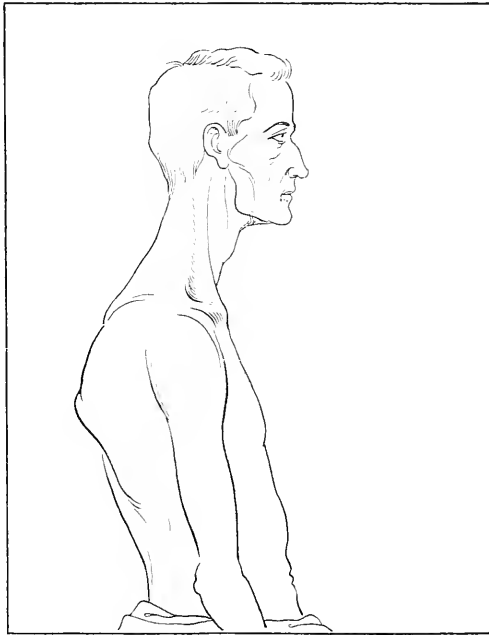


FIG. 66.—The phthisical or flat chest. Habitus phthisicus.

note a gain in weight during afebrile periods, and if the fever is mild during acute exacerbations and of short duration, the loss in weight may be insignificant. They may be ahead in this regard at the end of a year or two, although the process in the lungs remains stationary or has even progressed.

Toward the end the emaciation is very pronounced and deserves the name consumption. Then it is not only the fever, cough and expectoration that are exhausting the patient, but also the lack of nourishment owing to anorexia, diarrhea, and perhaps dysphagia when the larynx is implicated. The preservation of the body weight, which is very frequent in fibroid phthisis, is only rarely seen in chronic

phthisis, and when found it is an indication of improvement, or that the quiescent periods are of long duration.

Hemoptysis is comparatively infrequent during this period, excepting in very advanced cases with cavities, when a terminal hemorrhage may carry off the patient, and in those suffering from hemorrhagic phthisis (see p. 192) it may recur at irregular intervals. As was already stated, most of the hemorrhages at this period, even when profuse, end in recovery.

The other symptoms of chronic phthisis have already been described in detail in previous chapters.

**Physical Signs.**—Depending on alterations in the pulmonary parenchyma, pleura, mediastinum and chest walls, the physical signs of advanced phthisis are complex. By percussion and auscultation we may determine with a reasonable degree of certainty the nature of the lesion, as well as the condition of the apparently unaffected parts of the thoracic viscera. But with the progress of the disease, the changes found on physical exploration become more and more variegated and, owing to frequent overlapping of pathological changes, their complexity is so great that it is often quite difficult or impossible to determine exactly the details of these changes by physical examination. This is well illustrated by the difficulty of differentiating pleural adhesions before inducing a therapeutic pneumothorax, and by the number of cavities that are missed during life and found at necropsy. Radiography is of immense value at this stage, but it is not infallible, as has already been shown.

**Percussion.**—The tuberculous infiltration usually extends in horizontal planes, though frequently metastatic deposits of tubercle are found at a distance from the original focus in the same or the opposite lung. The result is that the impairment of resonance found over one apex during the incipient stage extends mainly downward, and, in progressive cases, we soon find dulness as far as the third or fourth rib or lower. The pitch of the note depends on the density of the infiltration, on the presence or absence of excavations and on the condition of the pleura. On the unaffected side a hyperresonant note may be elicited, which may be accentuated by vicarious emphysema.

Dulness is very frequently found in the interscapular spaces which may be an expression of enlarged peribronchial glands, or infiltration of the apex of the lower lobe of the lung. In the majority of cases there is more or less retraction of the base of the lung, easily made out by tidal percussion.

With percussion we may also determine the position of the heart which in many cases is of immense diagnostic significance, as has been pointed out elsewhere by the writer.<sup>1</sup> In phthisis the heart is, as a rule, dislocated toward the affected side, the reverse of conditions

<sup>1</sup> Arch. of Intern. Med., 1914, xiii, 656.

found in pleural effusions, pneumothorax, etc. It is therefore important to determine the position of the heart in cases showing intense dulness of the lower parts of the chest on one side when the problem arises whether it is due to an effusion, or to thickened pleura with pulmonary retraction. Exploratory puncture, if negative, is not conclusive, but when we find the heart displaced to the opposite side, we may conclude that there is an effusion, while when it is dislocated toward the affected side, it is due to excavation and to pleural thickening.

The routine methods of physical exploration show the location of the heart in phthisis easily and vividly; but in many cases the diagnosis is difficult, and occasionally almost impossible. The side of the heart adjoining the healthy lung is easily made out by percussion, but the cardiac dulness at the side adjoining the affected lung merges with the dulness of the infiltrated and consolidated lung tissue or thickened pleura, and it is difficult to separate by any method of percussion. The fluoroscope and the skiagraphic plate also fail at times to show a definite outline of the borders between the heart and lung. Indeed, I have found at times that orthodiagraphy was of no avail.

Dextrocardia is not rare in extensive right-sided lesions. It is to be differentiated from complete transposition of the viscera by the location of the liver, spleen, etc.

**Auscultation.**—Auscultation in advanced phthisis is of even greater diagnostic significance than percussion and skiagraphy, because it shows distinctly the progress of the process in the lungs, especially its activity. The diagnosis of a healed lesion can only be made by a study of the constitutional symptoms and a careful consideration of the auscultatory phenomena elicited over the chest.

The breath sounds which, during the incipient stage, may have been somewhat altered, rough, cog-wheel or feeble, now become more and more bronchial or tubular in character. Excepting in very acute cases, which do not concern us here, bronchial breathing does not appear suddenly in chronic phthisis. Following a progressive case we may observe that the cog-wheel breathing changes by degrees; first the expiratory murmur becomes prolonged, then the sounds assume a bronchovesicular character, indicating that the breath sounds are mixed, the vesicular coming from the healthy lung, and the bronchial from the disseminated infiltrated patches. When these patches conglomerate and the part of the lung consolidates into an extensive airless area, thus acting as a good conductor of the tracheobronchial murmur to the surface, we get bronchial breathing. With the onset of softening the products of tissue disintegration are expelled, leaving an excavation and we often, though not invariably, hear cavernous or amphoric breathing, which will be discussed later on.

The advance of the lesion is characterized pathologically by softening of lung tissue, followed by liquefaction and cavity formation.

These changes are best determined by auscultation and the detection of moist rales which are produced by the air current passing through the bronchi into the diseased area filled with débris of disintegrated tissue. These rales are of various sizes—large, medium or small—according to the size of the bronchus or excavation in which they are produced. Usually they are consonating, ringing and either provoked or intensified by cough. Their diagnostic significance lies mostly in their localization and persistence. They are mostly found over the suprascapular fossæ, in the upper part of the interscapular space and especially above and below the clavicle and with them we usually hear low-pitched, bronchial breathing. When heard unilaterally and persistently in any of these locations, they are, with but few exceptions, pathognomonic.

The onset of softening is characterized by the appearance of moist rales, usually small or of medium size. They have been called by the French *râles de friture* because they simulate the sounds heard when frying something. But we must guard against overestimating the extent of the disease by wide distribution of rales. With concomitant bronchitis they may be distributed all over the chest or all over one hemithorax, while the tuberculous lesion is rather limited. After pulmonary hemorrhages the rales are heard far away from the tuberculous area and we must be guarded in concluding that it is an indication of wide-spread extension of the tuberculous lesion. The thermometer is a better guide under such circumstances.

On the whole, it can be stated that the activity of the tuberculous process may be gauged by the number, character and distribution of moist rales audible over the chest. The larger their number, the larger their consonance, when localized over a limited area, the more active the process, while absence of rales, coupled with absence of constitutional symptoms, indicates an arrest in the progress of the disease.

Sibilation is quite frequently heard in cases of advanced phthisis and it may be caused by various conditions. In the interscapular spaces and near the two sides of the sternum whistling sounds are an indication of tracheobronchial adenopathy with pressure on the bronchi. In some cases, we hear sonorous rales all over the chest or unilaterally in cases complicating bronchitis or emphysema; over areas of localized vicarious emphysema, sibilation is also heard at times. For a long time or permanently, after a lesion has healed, there may remain sibilation, "cicatricial rales."

Friction sounds are very frequently heard. Their significance has already been discussed.

**Cavities.**—This stage is characterized by the formation of pulmonary excavations. The constitutional symptoms accompanying the formation of cavities depend on the acuteness of the process. As long as the excavation is surrounded by infiltrated and caseated lung tissue, the symptoms are acute—high fever of a continuous or remittent

type, profuse nightsweats, severe cough with abundant expectoration, rapidly progressing emaciation, etc. But in most cases the process is not so acute. The excavation is surrounded by a fibrous shell which limits its progress and prevents absorption of the toxic products to a great extent, so that the patient may feel quite well despite the formation of more or less extensive excavations in his lungs. In the chronic cases that do not succumb, but do not heal either, the cavity may keep on secreting mucopurulent matter which is promptly removed through the fistulous tract that leads to a bronchus.

It is in these chronic cavitory cases that we meet with the undulating clinical picture of phthisis described above. Whenever the fistulous tract leading from the cavity is obstructed, the amount of expectoration is diminished and fever, nightsweats, etc., result, till the plug in the bronchus is dislodged, when expectoration begins and the patient again feels comparatively well.

**Diagnosis of Cavity in the Lung.**—If we should accept the signs given in text-books as infallible criteria, the diagnosis of cavities is very simple. But those who often make autopsies and have opportunities to verify their findings are frequently amazed at the large number of cavities found *intra vitam*, but missing at the autopsy, and the reverse.

In order that a cavity should be discerned by physical exploration or even by skiagraphy, it must attain the size of at least four centimeters in diameter; it must be superficially located, filled with more air than secretions and communicate with a bronchus. In the apex cavities are often missed because the thick, indurated pleura screens all signs. Some even maintain that they must have smooth walls if we are to elicit by auscultation and percussion the signs which are characteristic of excavations. In fact, Walsh, Landis and others who have studied the physical signs of vomicae, verifying their findings at necropsies, found that many excavations are overlooked, while others that are diagnosed are not found at the autopsy. For this reason some believe that the presence of elastic tissue in the sputum is the best sign of pulmonary excavation.

Inspection and palpation are of little value. The muscular atrophy noted over deep excavations above and below the clavicle may be seen in pulmonary retraction without excavation. Over superficial cavities, extreme atrophy of muscles and integuments of the area overlying the excavations is very frequent. This atrophy leaves the chest wall over a circumscribed area very thin and, combined with pleural adhesions and retraction, may cause a cup-shaped depression localized over the site of the cavity. But this is comparatively rare, probably because many cavities are deeply situated within the lung.

Percussion over a cavity gives a dull note, and only over large excavations superficially located in the infraclavicular region of emaciated patients and filled mostly with air, may be obtained a hyperresonant or tympanitic note. At most, we usually find dulness with a tympanitic overnote. But to indicate excavation even this must be strictly



localized and circumscribed. The resonance may change within a single day from tympany to dulness when it fills up with secretions.

On the whole, cavitary tympany depends on many factors. In young persons with elastic and resilient chest walls it is more often present over small excavations than in the aged, whose chests are usually rigid and unyielding, and even large excavations may not be tympanitic. The more superficial the location, the more pronounced the tympany, while deeply lying cavities are screened by air containing lung tissue and tympany is altogether absent. It is thus evident that tympany is not a constant sign of cavitation, but when localized, circumscribed and pronounced, it speaks for a cavity of large size with greatly relaxed walls; and conversely we find high tympany over tight walls of small cavities. It may best be perceived, as Flint showed long ago, when the ear is close to the patient's mouth, or when the bell of the stethoscope is held in this position. Cracked-pot resonance is also best perceived in this manner.

The most common site of tympany due to cavitation is above the fourth rib anteriorly and on rare occasions we find it in the axillary line beneath the fifth rib, especially in the left side, while posteriorly it is exceedingly rare because of the large muscles which interfere with percussion. I have met with cavities that were tympanitic over three-fourths of the chest wall, indicating excavation of almost an entire lung. But this is rare because in such cases the mediastinum is pulled over and produces dulness.

Occasionally the tone changes known as Wintrich's, Friedreich's and Gerhardt's phenomena are of assistance in the diagnosis of vomicae, but not as frequently as some text-books would lead us to believe.

*Wintrich's phenomenon*, obtained by percussion while the patient opens and closes his mouth, the note being tympanitic when it is open, and of lower and deeper pitch when closed, is a good indication of a cavity communicating with a bronchus and is more distinct, the greater the diameter of the bronchus. It may be obtainable only in certain positions of the body (interrupted Wintrich), which is clearly due to the presence of fluid secretions within the cavity which obstruct the opening of the bronchus. It is also met with in some cases of bronchiectatic excavations, but this is to be distinguished by the location of the cavity—anteriorly and above in tuberculosis, and posteriorly and below in bronchiectasis. It may also be found in pneumothorax, but the concomitant symptoms and signs clear up the diagnosis, excepting in the localized and latent forms, which can only be recognized with the *x*-rays.

*Williams's tracheal tone*, observed while percussing the consolidated apex which conducts the tracheal tympany, is at times mistaken for Wintrich's phenomenon. It is usually found in cases of contraction or consolidation of lung tissue or its compression in pleuritic exudates, when percussion above and below the clavicle sets up vibrations in the main bronchus and the trachea.

*Friedreich's phenomenon* consists in high-pitched tympany over the site of excavations when the patient holds his breath during full inspira-

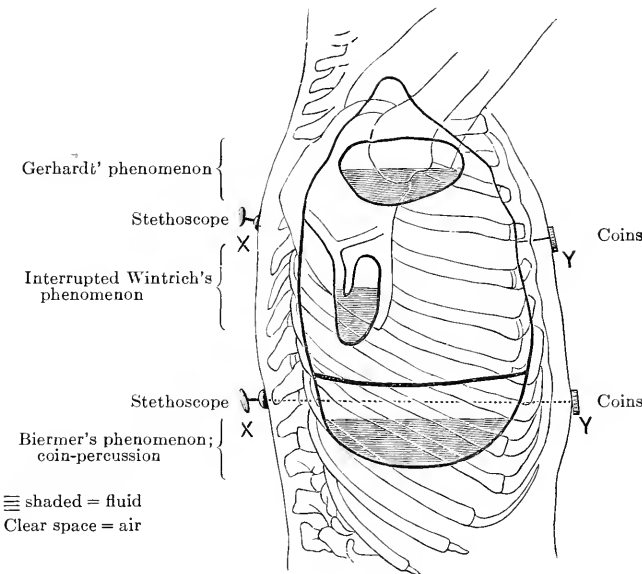


FIG. 67.—Illustrating Gerhardt's and Biermer's phenomena, interrupted Wintrich's phenomenon and coin-percussion. (Musser.)

tion, diminishing during extreme and held expiration. This is not as reliable as Wintrich's sign because it is at times obtained over healthy lungs.

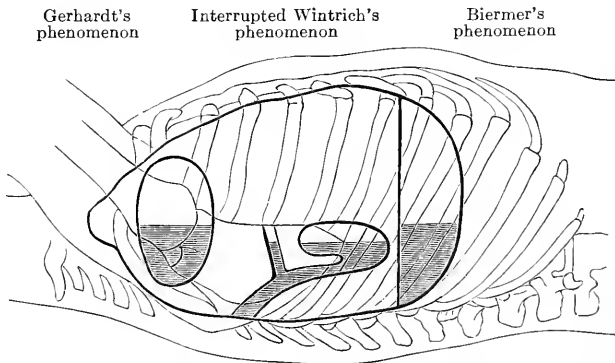


FIG. 68.—Illustrating Gerhardt's and Biermer's phenomena and interrupted Wintrich's phenomenon. (Musser.)

In *Gerhardt's phenomenon* the note is higher and more tympanitic when the patient is sitting or standing than when he is reclining, and is said to be characteristic of an oval-shaped cavity filled partly

with fluid and partly with air, the fluid gravitating according to the position of the patient. Small cavities, superficially located, occasionally show this sign and when the excavation is centrally located, it must attain considerable dimensions to be thus characterized. As Sahli points out, Gerhardt's phenomenon is rare, and slight differences in the percussion note with changes in position may be within physiologic limits due simply to alteration in the tension of the thoracic walls without any cavity within the chest.

In hydropneumothorax we often observe *Biermer's phenomenon*, which is produced in the same manner as Gerhardt's in pulmonary cavities (see Figs. 67 and 68).

**Cracked-pot resonance**, first described by Laennec, is occasionally obtained over cavities. Some precautions are necessary in order to elicit this sign. The patient should keep his mouth wide open, the pleximeter finger placed over the second or third intercostal space anteriorly, and with the percussion finger a strong blow is delivered without rebound, at the end of expiration. It is apparently a stenotic murmur at the opening of the cavity into a bronchus when the air is suddenly expelled through a narrow, slit-like opening. It may, however, be met with in many other conditions, as in a crying child, and in adults with relaxed lungs, also in emaciated persons with resilient chest walls and in cases of small emphysematous islands surrounded by consolidated lung tissue which are not uncommon in chronic phthisis. Of the many cavities that I have seen, cracked-pot resonance was present in but a small proportion. When obtained in connection with some of the other signs, it is of significance.

**Cavernous and Amphoric Breathing.**—Auscultation may be altogether negative over deeply lying vomicae, or such as are completely closed by a plug in the communicating bronchus. Cavernous breathing is often heard; it resembles the sound produced while blowing into an inclosed hollow space. It is caused by the overtones developed in the cavity by reflection from the walls. Over cavities having smooth walls communicating with a bronchus we often hear amphoric breathing—a murmur with high overtones lacking deep basal tones, resembling the sound produced by blowing across the opening of a narrow-mouthed vase. Cavernous and amphoric breathing have a certain diagnostic significance. They indicate pulmonary excavation, bronchiectasis, or pneumothorax. Formerly it was thought that pneumothorax shows amphoric breathing only when it is freely communicating with a bronchus. But now we often find it over artificial pneumothorax, and it is then due to reverberation of the bronchial sounds from the smooth pleura. Over many excavations only loud and harsh bronchial breathing is audible.

Over areas with amphoric breathing we usually elicit a dull note on percussion and, at times, cracked-pot resonance, while over areas with cavernous breathing we often get tympanitic resonance, though not always, as was already indicated. Amphoric resonance is an

indication that the excavation is at least five centimeters in diameter, that its walls are smooth, round and rigid due to surrounding infiltration or fibrosis; that in all probabilities it communicates with a bronchus of not very wide caliber; and that it is not active—a fibrous capsule prevents the absorption of toxic matter from the cavity, and also the extension of the lesion, and the small amount of secretion is soon eliminated by expectoration. It is for these reasons that cavities with amphoric breathing are usually not accompanied by any adventitious sounds, excepting at times by a metallic tinkle, and this is very rare; while cavernous breathing is almost always accompanied by large or medium sized, consonating rales or gurgles. In the latter case the cavity is active, probably growing and not surrounded by a fibrous shell. The prognostic significance is clear. The intensity of the amphoric phenomena depends on the stiffness of the wall which, in its turn, depends on a strong fibrous capsule or on infiltration and caseation of the surrounding lung tissue. In the former case it will not enlarge and may even shrink, while in the latter case the excavation may extend and usually does.

**Metamorphosed Breathing.**—Over the sites of cavities, mainly over the upper lobes, we sometimes hear the inspiratory murmur begin as a harsh or bronchial murmur, but during its course suddenly soften and change in tone, finally ending with an amphoric sound. At times both inspiration and expiration are thus affected. Laennec spoke of it long ago as a *soufle voilé*, beginning as vesicular and ending as bronchial or amphoric. It seems that it is due to the breathing of a cavity. The air enters into a relaxed excavation and the murmur is modified while its walls are being distended or inflated. It is one of the best signs of an excavation, but it is only rarely met with.

**Adventitious Sounds Heard over Cavities.**—Over excavations, large, moist, bubbling, consonating, rales—called in text-books metallic or cavernous rales—are often heard. They are caused by the air stream passing through the collection of fluid in the excavation. The size, pitch, timber and duration of these rales depend on the size of the vomiceæ in which they originate as well as the condition of its walls—whether they are smooth or ragged, rigid or relaxed, etc. On the other hand, over old cavities there may be audible amphoric breathing of an exquisite type, metallic breathing without any rales at all, because the fibrous walls do not secrete any more. These are cases that are doing well for years in spite of extensive excavations.

In many cases the number of rales in excavations, and their intensity are so great that they obscure all the breath sounds.

**The metallic tinkle** is only rarely heard over pulmonary cavities.

**Pectoriloquy** is met with over pulmonary cavities, but it is not pathognomonic of this condition. In many cases we hear the voice as if it is directly spoken into the ear with abnormal clearness. It merely indicates that the conditions for conduction are unusually good, which may be true of excavations, but is also met with in

pneumothorax and even in consolidated lung tissue through which a bronchus passes.

The same is true of whispered pectoriloquy. But the transmission of the whispered voice with a metallic or amphoric echo, which Kuthy calls "amphorophony," is a sure indication of a smooth-walled cavity filled with air, either pulmonary or pleural, *i. e.*, a tuberculous excavation or a pneumothorax. The differential diagnosis between these two conditions can at times only be made out by the *x*-rays, and I have met with cases in which skiagraphy was not decisive. Some cavities can be made out by auscultation with much less trouble and greater reliability than with other diagnostic methods. Amphorophony is, however, only audible over old and large cavities which are stationary, while over acutely progressive and extending vomicea it is not at all heard.

**Basal Cavities.**—The vast majority of tuberculous cavities are formed in the upper lobes of the lungs, except in the terminal stages, when the resistance is very low, excavations then forming in the lower lobes of the lungs.

They are very difficult of diagnosis. We may find signs of excavations at the base which are really "phantom caverns," as William Ewart<sup>1</sup> called them. The amphoric sounds of an excavation in the upper lobe are transmitted to the base by some transient or permanent consolidation. Echo may also be responsible for cavernous sounds at the base when the original excavation is situated in the opposite side of the chest and not in immediate contact with the spinal column.

Basal cavities are to be differentiated from bronchiectasis and from syphilis of the lungs. In bronchiectasis the sputum is mucopurulent, separates into three layers on standing, is occasionally putrid, brought up periodically in large quantities and contains no tubercle bacilli. But all these may be encountered with phthisical cavities. The writer has been guided by the state of nutrition of the patient. If, in spite of the abundant and extensive bronchitis manifesting itself by profuse expectoration and numerous large, consonating rales and gurgles, the patient holds his own, the chances in favor of bronchiectasis are immense. Tuberculosis showing such activity is accompanied by pronounced emaciation, fever, night sweats, and tubercle bacilli are not lacking. Syphilis of the lung with basal cavities is differentiated from tuberculosis by the presence of other stigmata of specific disease, the Wassermann reaction, and the continued absence of tubercle bacilli from the sputum. Finally, the diagnosis is at times only cleared up by the therapeutic test—antisyphilitic treatment acts promptly in most cases.

It is important to mention that the prognosis is more unfavorable in basal cavities than in those located in the upper lobes, undoubtedly because they do not empty themselves with ease. Considering a

<sup>1</sup> Goulstonian Lectures, Brit. Med. Jour., 1882.

pulmonary cavity as an abscess, we understand that when it does not drain the result must be disastrous; the abundant secretions fill it up and cough is not very effective in removing them. In the terminal stages of phthisis with lesions in the upper lobe, excavations sometimes form at the base, as we find them at necropsy, and kill the patient who may have been getting along very well before their occurrence. In fact, if in the course of chronic phthisis signs of excavation appear in the lower half of the chest, the prognosis is very gloomy.

**Visceral Displacements.**—The displacements of the mediastinal organs have already been referred to (p. 335). The heart is in most cases of advanced phthisis displaced toward the affected side of the chest and in right-sided lesions we at times meet with complete dextrocardia. But in many cases there are also to be noted displacements of the trachea and larynx, first described by E. Ruedinger.<sup>1</sup> More recently Gerald B. Webb, A. M. Forster, and G. B. Gilbert<sup>2</sup> described in detail the tracheal position in phthisis and suggested an easy method of detecting it: By placing the hand behind the neck while the thumb anteriorly reaches out to the trachea and rolls it, we can in most cases determine its position. It appears that in most cases of early phthisis the trachea is displaced toward the affected side. Webb found in 100 cases of pulmonary tuberculosis of all stages the recognition of the side especially affected proved correct in 69, doubtful in 19, and incorrect in 12 cases. It is due to pleural adhesions, together with fibrosis in the lung or pulmonary retraction pulling the trachea along.

This deviated trachea is occasionally a source of error in diagnosis. When it is displaced to the margin of the sternum, we hear loud tracheal, or even "cavernous" breath sounds both anteriorly and posteriorly and thus diagnose a cavity which does not exist. Especially is this error of great moment when the trachea is displaced to the opposite unaffected side after the induction of a pneumothorax, and we may think that there is a cavity in the untreated lung. But a little care will usually clear up the case, especially when the possibility of displacement of the trachea is borne in mind. Webb says that movement of the trachea to the side of the healthier lung following the application of pneumothorax foretells a successful application of this procedure. In my experience this is not invariably the case.

In many cases there is also upward displacement of the stomach and liver after pulmonary retraction.

**Duration of the Disease.**—The duration of chronic phthisis is variable. Some patients get well or succumb within one year, while in most the sluggish course continues intermittently for many years, during which period the patients consider themselves cured, and suffer from "relapses" several times. They constitute the bulk of the class of patients who are admitted to sanatoriums and hospitals for consumptives several times. The reason is clear when we bear in

<sup>1</sup> Beitr. z. Klinik d. Tuberkulose, 1910, xvii, 151.

<sup>2</sup> Jour. Amer. Med. Assn., 1915, lxx, 1017.

mind the oscillating course of the disease—during acute or subacute exacerbations they seek relief in an institution, while during remissions, when the process is quiescent, they believe that they have been cured, or the disease has been arrested.

Basing their estimates on heterogeneous material, different authors have estimated the average life of the consumptive as at from one to ten years. Leudet<sup>1</sup> found that of hospital patients 90.7 per cent. die within five years of the onset of the first symptoms; 9.3 per cent. during the sixth to the nineteenth year. He also found that among the more prosperous patients only 77.2 per cent. die within the first five years, and 22.8 per cent. between the sixth and the nineteenth years. Brown and Pope,<sup>2</sup> studying statistically the outlook of patients discharged from the Adirondack Cottage Sanitarium, found that, of those discharged “apparently cured” at the end of five years, 94 per cent. of the expected were alive; at the end of ten years, 86 per cent. In those “arrested” the proportions for the corresponding years were 63, 49, and 46 per cent.; and for those “active,” 25, 15, and 10 per cent. It is thus clear that “an arrested” or even an “active” case is not necessarily doomed. There are always good chances to live for long years.

The striking disparity in these two sets of statistics is due to the difference in the material. Leudet studied only fatal hospital cases, without including any of those who survived twenty years, while Brown and Pope studied cases discharged from a good sanatorium in which moderately well-to-do patients predominate, and among whom a fairly large proportion were affected with the abortive type of the disease.

Attempts at estimating the average duration of life of the consumptive have also met with failure because it is difficult to obtain comparable material. When only acute, progressive cases are considered, the average is a low figure, one year or even less; when abortive cases are considered—and they are mostly those which have been diagnosed exceedingly early in the disease—the average is very high. It is for this reason that the estimates of “averages” vary from one to ten years, according to different authors.

But for the individual patient, with whom the physician deals, averages do not count for much. He must be judged by the clinical manifestations. It may be stated that those who have long periods of quiescence live long; many practically their natural life. They may be “cured” several times when they suffer from acute or subacute exacerbations, but they recuperate every time and live on, often with quite some efficiency. On the other hand, those in whom acute or subacute exacerbations are frequent, and each is of long duration, a fatal issue is inevitable sooner or later.

<sup>1</sup> Quoted from Kuthy and Wolff-Eisner, *Prognosenstellung d. Tuberkulose*, Berlin, 1914, p. 56.

<sup>2</sup> *American Medicine*, 1904, viii, 879; *Ztschr. f. Tuberkulose*, 1908, xii, 205.

**Modes of Death.**—Death supervening during an acute exacerbation, when the process in the lungs is extending, or the toxemia is severe, or the resistance is low, may be rapid, like from pneumonia or septicemia. The patient may have done quite well, but is suddenly stricken with high fever and prostration, and he succumbs to dyspnea, cardiac failure, etc. Usually the process is slower; the high continuous or remittent fever, the profuse night sweats, anorexia, dysphagia due to laryngeal ulceration, extreme emaciation, etc., keep on for weeks or months; the patient is gradually but surely consumed by the disease. In some, the last few weeks resemble in their symptomatology the typhoid state with marked prostration, muttering delirium, etc.

In others, the cachexia progresses despite the fact that the fever is low, hardly ever exceeding 101° F., and the patients finally die from asthenia, like those suffering from malignant disease. Excepting the cough, diarrhea and weakness they do not suffer much and, because the sensorium is well retained to the end, the euphoria may be exquisite. Others consider themselves quite well despite the extreme emaciation and attempt to walk around, against the advice of their physician and among them death due to syncope may occur. Some of these unfortunates are occasionally found dead in bed in the morning. But in such cases it was usually not syncope but a heavy dose of some opiate which abolished the reflexes, prevented cough and expectoration, and they were drowned by their own secretions. Other causes of sudden death during the night are spontaneous pneumothorax, copious hemorrhage, etc., killing before aid can be summoned.

Complications of the disease are often responsible for a fatal issue. Among the most important are pulmonary hemorrhage and pneumothorax. While 98 per cent. of patients who suffer from more or less bleeding survive the accident, 2 per cent. succumb to it. The patient may feel comparatively well, and in fact consider himself on the way of recovery or even cured, when suddenly brisk and profuse hemorrhage occurs and kills him. Emaciated patients may die as a result of suffocation with their own blood, being powerless to expel it from the chest. In others, the hemorrhage may not be fatal, but it is instrumental in spreading the process in the lung, causing bronchopneumonia, which is fatal in a few days or weeks.

Pneumothorax is the cause of death in about one of 150 fatal cases of phthisis. This may kill the patient within one or two days, the cause of death being asphyxia, or within a few weeks or months through complicating pyothorax.

Complicating laryngeal tuberculosis is responsible for the death of many patients through dysphagia, dyspnea, edema of the glottis, etc.

Between 5 and 10 per cent. of deaths from phthisis are preceded by cerebral symptoms. Most of these are due to tuberculous meningitis, but some are also caused by uremia, as was already stated.

**Premonitory Signs of Death.**—In chronic phthisis with tendencies to a fatal issue, it is often very difficult to prognosticate the time



when the end will come. Indeed, the more extensive the experience of a physician with this disease, the more guarded he becomes in foretelling the day of death. Such statements as "he cannot survive three days," or "he will surely die within a week," etc., should be avoided. Some patients keep on living for weeks or months under conditions which are puzzling, to say the least.

There are symptoms and signs which may, however, be considered precursors of death in phthisis. Of these we may mention: Dysphagia, due to laryngeal ulceration, when not quickly relieved by treatment, is a sure indication that the patient will not survive very long. The same is true of profuse diarrhea which cannot be controlled by treatment. The emaciation is extreme and the end comes rapidly. But I have seen cases with profuse diarrhea lasting for months in spite of the fact that they hardly assimilated any nourishment. The reason is clear when we consider that the emaciated victim of phthisis lies quietly, hardly moving a limb or expending any energy, so that the least fuel is sufficient to keep the spark of life aglow.

Edema of the extremities very often appears shortly before death. It is usually due to cardiac weakness or nephritis, thrombosis or thrombophlebitis. It may be unilateral, but usually both lower extremities are affected. The swelling may be enormous in extreme cases, while in most it is but moderate, and tender on pressure. When this edema of the lower extremities is combined with cyanosis and dyspnea, a fatal issue may be expected within a month. Thrombosis of the femoral, jugular, subclavian, or other veins is one of the surest premonitory signs of death.

## CHAPTER XXI.

### ACUTE PHTHISIS.

JUST as in other infectious diseases, there are observed in tuberculosis acute, malignant or fulminating forms which run a shorter and almost invariably fatal course. They are relatively rare, as malignant scarlet, measles, typhoid, etc., are rare. Every practitioner meets with these acute cases and the laity is well aware of their existence. When tuberculosis makes its appearance in a member of a family anxious inquiries are made to ascertain whether it is not "hasty," or "galloping consumption," the names under which acute tuberculosis is commonly known. Pathologically, the lesion is practically the same as that of the chronic forms of the disease, considering that there are no two cases of phthisis in which the anatomical changes are exactly alike, but clinically it manifests itself by a more rapid course, the patient lasting as many months with the acute form, as years with the chronic forms. Acute tuberculosis may be said to be active chronic phthisis without the remissions and ameliorations characteristic of the course of the latter affection.

It is unnecessary to enter into hair splitting distinctions of the pathological and clinical types of acute phthisis described by some authors, notably the French. In practice we meet mainly with two types of the disease: The lobar pneumonic type—acute pneumonic phthisis, and the lobular or bronchopneumonic type. In the former the patients are usually adults, while the latter attacks mainly infants and very young children, and adults only at the terminal stages of chronic phthisis.

Between the two extremes—chronic and acute phthisis—there are many gradations, some are very acute, the patient being carried off within one or two weeks; some are subacute, lasting for two to four months, others even a year, but without any remissions in the progress. Then there are acute exacerbations during the course of chronic phthisis which are anatomically and clinically of the same character as the acute or subacute forms and often bring hitherto hopeful cases to a speedy termination. I have also met with cases which began acutely and kept up in that manner for several weeks, but suddenly or by degrees took a turn to the better and the patient passed through the course of chronic phthisis subsequently.

**Etiology.**—The factors operative in causing an acute and malignant evolution of phthisis in some cases, while in the vast majority it is chronic, slow and more or less benign, are not clear. From a careful

study of the cases met in practice it appears that the general condition of the patient before the onset of the disease has no influence in this direction. In fact, it appears, as was already stated (see p. 112), that phthisis in those who suffered from scrofula during childhood, or who are descended from tuberculous stock, is more likely to run a slow, sluggish course. On the other hand, we very often meet with acute phthisis in persons who have no hereditary taint, who have been in excellent condition, and only rarely in the weakly and decrepit, excepting tuberculous bronchopneumonia in infants.

The problem whether these acute cases are invariably due to more virulent strains of tubercle bacilli has not been solved, though there appears to be no evidence in favor of such a view. Some authors have held that acute phthisis is caused when a tuberculous cavity breaks through into the lung, disseminating the secretions containing bacilli, but this is negated by the fact that we meet numerous patients who never coughed before the onset of the acute disease.

It appears that individuals who have never before been in tubercle laden surroundings are more likely to develop acute phthisis when infected primarily after they have passed the age of childhood, as we have already shown (see p. 119). The same "virgin soil" is presented by infants: when they are infected with tuberculosis they very often suffer from the acute forms of the disease, and so do adults hailing from rural districts where they have not met with tuberculosis, so that if infection takes place it is primary. The explanation of these phenomena has been discussed in a previous chapter.

**Acute Pneumonic Phthisis.**—The anatomical changes are those of pulmonary tuberculosis but the process of caseation and liquefaction gains the upper hand, not being limited by the conservative process of fibrosis which is a strong feature in chronic phthisis; no connective tissue is formed to localize the lesion. Usually the greater part of a lobe, or a whole lobe, is affected. The parenchyma is transformed into a solid, caseous or gelatinous mass within which there can often be found a focus representing an old lesion. The destruction of lung tissue goes on at a rapid pace and within a short time more or less extensive excavations may be formed. But these excavations are not surrounded by a connective-tissue wall; all around them is caseated lung tissue. In many cases, however, death supervenes before softening has had time to set in and sequestrate the affected part of the lung. We may find scattered tubercles or caseous nodules all over the affected lung and also in the other, as well as on the visceral pleura, but pleural adhesions are extremely rare.

**Symptomatology.**—The disease is mostly seen in adults between twenty and forty years of age. The onset and symptoms during the first few days are akin to those of lobar pneumonia. In fact, most of the cases of chronic phthisis which are said to have begun as lobar pneumonia are cases of acute pneumonic phthisis which were not recognized as such at the onset of the acute stage.

As given by the patients, the onset is nearly always acute. After some alleged exposure there was a chill, fever, pain in the chest, cough, etc. But a careful inquiry elicits that while the acute symptoms have come on suddenly, the patient has for weeks, perhaps for months, felt out of sorts; was unable to perform his usual work without fatigue; in fact, he has coughed, expectorated and may have had some night-sweats. But all these symptoms were not sufficiently pronounced to cause alarm; even if he has consulted his physician he may have been told that his troubles were trifling. This long prodromal stage is of great diagnostic importance, and will often aid while attempting to differentiate acute pneumonic phthisis from lobar pneumonia.

With the acute symptoms the patient is laid up in bed. The dyspnea is marked from the beginning and may be paroxysmal. The pain in the side is mild and only rarely as acute as in pneumonia or pleurisy, or may be altogether lacking. Cough is nearly always annoying; it may be severe, incessant and exhausting. At first dry, it slowly becomes productive and the sputum is at times rusty and viscid, adhering to the sides of the vessel like in lobar pneumonia. But in most cases it is mucopurulent, frothy and easily brought up. In some cases it is sanguineous, at times repeated, small, true hemoptyses take place, and the disease may begin with a profuse pulmonary hemorrhage. When softening and excavation take place, which occur quite soon, the sputum is of the same character as that of chronic phthisis, excepting that it is more often green in color. In the beginning repeated microscopic examinations do not reveal any tubercle bacilli, and because pneumococci are quite frequent, the diagnosis is very difficult. Only after the disease has lasted for a couple of weeks, and very often much later, and we may be thinking that we are dealing with an unresolved pneumonia, tubercle bacilli are discovered in the sputum.

Weakness, anorexia, emaciation and fever are very strong clinical features in the evolution of the disease. The weakness may be so severe that very early in the course of the disease the patient is unable to sit up in bed, or to breathe for the purpose of auscultation. When examined they fall back in bed exhausted, pale and cyanosed. This asthenia is not seen in the average case of lobar pneumonia. With the anorexia, which may be pronounced from the very beginning, emaciation goes hand in hand. Even in the few cases in which the appetite is somewhat retained, the emaciation is very early and pronounced, and out of proportion to the fever and anorexia. It usually proceeds rapidly and often frightfully, so that within a few weeks a normally built man is reduced to a skeleton. Wasting is particularly quick in the muscles of the chest.

In the beginning the fever is of a continuous type, like in lobar pneumonia, though some authors have described pneumonic phthisis without high fever, which I have never met in my practice. But this is rare during the first few weeks when the temperature curve ex-

quisitely simulates that of lobar pneumonia, but during the second week, when we expect defervescence, we are disappointed. Instead of this, the fever becomes intermittent or hectic, with morning remissions to normal or even below, and afternoon rises to 103° or 104° F., and accompanied by copious night sweats. The pulse is rapid, small and feeble, and the blood-pressure low. The full, vigorous pulse of lobar pneumonia is never found.

**Physical Signs.**—Physical exploration of the chest often shows the signs of typical lobar pneumonia. There is impaired resonance or dullness over the upper part of one side of the chest above the third rib. But instead of the harsh tubular breathing which is characteristic of pneumonia, we usually perceive diminished and, in some cases, complete absence of breath sounds, which are replaced by moist, subcrepitant rales. The crepitation of pneumonia is only rarely audible. With the advance of the lesion the dullness becomes more pronounced and the respiratory murmur may be altogether abolished, or bronchial breathing may become audible coupled with small and medium-sized moist rales. In acutely progressive cases signs of excavation may be found within four weeks, but this is rare.

**Course.**—In most cases the acute symptoms persist for two or three months, the lesion softens, extensive excavations may form and the patient finally succumbs to asthenia. In some the process is of shorter duration; I have seen two cases in which death occurred in less than three weeks. On rare occasions the disease is acute for four to six weeks, when an improvement in the general condition takes place and, with more or less extensive excavation in a lung, the patient becomes a chronic consumptive and the disease may even be arrested in time, which is, however, very rare. In some the toxemia is very severe and the patient succumbs within two or three weeks, even before softening has taken place. The prognosis under the circumstances is very grave, the average duration of the fatal cases, and they are in the vast majority, is about six weeks, dying from toxemia and exhaustion.

**Differential Diagnosis.**—It is often very difficult to differentiate acute pneumonic phthisis from lobar pneumonia, especially during the first two weeks of the ailment. Mistakes may be avoided by carefully inquiring for premonitory symptoms of tuberculosis preceding the acute onset, such as anorexia, emaciation, weakness, mild cough, night sweats, etc., which are frequent in acute phthisis, while in lobar pneumonia the patient is stricken suddenly when he feels in the best of health. In fact, in atypical pneumonia, acute tuberculosis is always to be thought of. The absence of pain in the side, the late arrival of true bronchial breathing, the hemoptysis, etc., may all lead to a diagnosis or at least a suspicion of acute phthisis. An irregular temperature curve, mild dyspnea, severe pallor, low leukocyte count, absence of pneumococci from the sputum and a strong diazo-reaction may also be considered. Of great importance in favor of acute phthisis is yellow or green sputum. Tubercle bacilli are conclusive

evidence, but they are only rarely found before the end of a month. During the first week the emaciation is negligible in pneumonia irrespective of the acuteness of the symptoms, while in phthisis it is immediately pronounced; night sweats, weakness and edema of the lower limbs are frequent. The crisis, which is sure to come before the fourteenth day in the vast majority of cases of pneumonia will clear up doubtful cases.

Especially difficult is the diagnosis of pneumonic phthisis in aged persons in whom it may occur without much fever and other general symptoms and only positive sputum can decide.

### TUBERCULOUS BRONCHOPNEUMONIA. GALLOPING CONSUMPTION.

**Etiology.**—The anatomical changes in tuberculous bronchopneumonia are those of pulmonary tuberculosis, excepting that the lesion is not localized in one apex, or one lobe, but disseminated all over one or both lungs in which there are distributed caseous nodules which vary in size from that of a pin-point to that of a walnut. Some authors have been inclined to attribute the wide dissemination of the lesion, as well as the acute course of this form of tuberculosis, to mixed infection with tubercle bacilli and pyogenic microorganisms. This, they believe, is confirmed by the fact that it very often follows infections such as measles, whooping-cough, influenza, typhoid, etc., showing that the patient had harbored a tuberculous process before, but with the addition of a new infective agent his vitality was reduced and the tuberculous process allowed to spread all over the lungs. But against this view may be brought forward the numerous cases in which mixed infection can be positively excluded.

In most cases it appears to be the result of the wide dissemination of the contents of a tuberculous cavity in the lungs, or the perforation of a tuberculous lymph node, the contents of which are aspirated, carried all over the bronchial tree and take root in various parts of the lungs. In infants, among whom this form of the disease is very common, it may be due to a primary massive infection with tubercle bacilli; the body possessing no immunity through previous infection, the result is the same as when a guinea-pig is infected. In adults, we also meet it after copious pulmonary hemorrhages, childbirth, in tuberculosis with diabetes and alcoholism, etc., when the resisting powers are at low ebb, and immunity acquired by the existing lesion is lacking.

**Symptoms.**—Tuberculous bronchopneumonia in adults is usually found in patients who have been tuberculous for some time. In those in whom it appears to be of sudden onset, careful inquiry elicits the information that the patient has been ailing for some time with symptoms highly suggestive of tuberculosis. In fact, it is often a complication of chronic phthisis: A patient who has been doing

fairly well suddenly develops acute symptoms without any special cause; more often after a profuse pulmonary hemorrhage or a surgical operation in which a general anesthetic was employed. Tuberculous women are frequently the victims soon after childbirth.

The clinical picture is that of an acute infectious disease with pronounced toxemia. The onset is sudden, often with a chill, fever, backache, cough, expectoration, etc. The fever is usually high—103° to 104° F. is not uncommon—and in children it may be even higher. The temperature curve is not characteristic; in fact, it may be stated that its characteristic is its irregularity. In many cases it is continuous with slight remissions, but in others it is intermittent, with chills before each rise. During the terminal stages it is usually hectic. The sweats are profuse and exhausting, the pulse feeble, small and rapid, 120 to 150 is not rare; the dyspnea is marked—40 to 60 per minute are very often counted and cyanosis is a frequent feature. Graves spoke of "acute tubercular asphyxia."

The intensity of the cough is variable: In some patients it is severe, painful, paroxysmal and may provoke vomiting. While occasionally the cough is mild, in most cases it is more severe than in chronic phthisis. At times expectoration is absent or scanty, but usually it is more or less abundant, often purulent, and, with the advance of the disease, nummular, yellowish green balls are brought up. Tubercle bacilli are found in most cases.

Hemoptysis is frequent in adults and may be quite copious; many cases begin with pulmonary hemorrhage.

The appetite is rarely fairly well retained, but in most cases this, as well as the digestive functions, are impaired, many have to be coaxed to take some nourishment. Emaciation proceeds at a rapid pace. Because of the flushed face it is at times not appreciated at first sight, but when the bedclothes are removed, the marked wasting of the subcutaneous tissues and muscles of the chest and extremities presents a frightful picture, especially when it is considered that it may have been consummated within a few weeks.

**Physical Signs.**—The physical signs vary according to the nature of the anatomical changes in the lungs. In the beginning they may be obscure and misleading. In most cases the note elicited on percussion is hyperresonant all over the two sides of the thorax; localized dulness is found only later when some of the disseminated tubercles have become confluent. Auscultation shows either feeble breathing or harsh bronchovesicular breath sounds all over the chest coupled with sibilant and sonorous rales. With the advance of the disease, which may be within but one or two weeks, we find localized areas, not necessarily in the apex, especially in children, of consolidation with bronchial breathing and moist subcrepitant rales which soon change their character when excavation takes place and the usual signs of a cavity can be made out. In many cases, notably in children, signs of diffuse bronchitis are found all over the chest, while in others

the toxemia is so severe that the patient succumbs before definite changes in the resonance and breath sounds have developed.

**Complications.**—Among these may be mentioned pulmonary hemorrhage, which may be fatal; intestinal tuberculosis, tuberculous meningitis and general miliary tuberculosis.

**Diagnosis.**—The diagnosis is very difficult in the initial stages particularly in children among whom it must be differentiated from postgrippal bronchopneumonia and sputum is not available for microscopic examination. In adults it is usually more easily diagnosed. We find in patients who have been tuberculous for some time that after a hemorrhage, surgical anesthesia, pregnancy, etc., the symptoms suddenly take a sharp turn and galloping consumption follows. It is always to be borne in mind that when in a person who never before had emphysema, and who has no barrel-shaped chest, symptoms and signs of emphysema suddenly make their appearance accompanied by acute constitutional symptoms such as fever, cough, night sweats, etc., acute phthisis is to be thought of. The sputum will soon clear up the diagnosis. With the advance of the disease the physical signs are easily made out.

**Prognosis.**—The prognosis is very grave. Some acute cases run a rapid course terminating fatally within four or six weeks, and in children in a shorter time. Many cases linger for three or four months and die of asthenia. I have met some cases in which the disease came to a halt and assumed the character of chronic phthisis.



## CHAPTER XXII.

### FIBROID PHTHISIS.

**Fibrous Hyperplasia in Phthisis.**—Discussing the morbid anatomy of phthisis, we showed that while the tuberculous process is mainly one of destruction—infiltration, caseation and softening—there are reparative forces at work in almost every case, manifesting themselves principally in the formation of connective tissue which either heals the lesion through cicatrization, or at least limits its progress. In fact, it may be said that without the formation of connective tissue, every case of phthisis would be acute. The balance between the destructive and reparative processes in phthisis depends consequently on the amount of fibrosis within and about the lesion—the more intense the formation of fibrous tissue, the slower the progress of the disease and conversely the less the fibrosis the more acute and progressive the disease.

We must distinguish between fibrosis and formation of cicatrices. When a lesion cicatrizes, the activity of the tuberculous focus is extinguished, though without any *restitutio ad integrum*, as is seen in healed tuberculous lesions of the lungs and pleura. But in fibrosis the lesion is an active, inflammatory process, though it may be only slightly progressive, yet connective tissue is being continually produced. In other words, in fibroid phthisis the destructive process is smouldering though in abeyance, or entirely absent and the proliferative process dominates. As Bard says, the lesions may be progressive and spreading, though they are not of a destructive character.

It must also not be confused with fibroid degeneration of the pulmonary parenchyma which at times follows acute or chronic non-tuberculous inflammatory processes of the lungs, such as the so-called interstitial pneumonia, pulmonary induration or cirrhosis, etc. Fibroid phthisis is a specific proliferation of the lung tissue caused by tubercle bacilli.

Clinically this form of tuberculosis is characterized by an exceedingly chronic course extending over many years, finally leading in most cases to the development of the symptoms and course of the common form of chronic phthisis. It differs from other forms of inflammatory fibrous degenerations of the lung in that it is caused by the tubercle bacilli and that the characteristic tuberculous giant cells are found microscopically in the lesions of fibroid phthisis.

Fibroid phthisis was mentioned by Bayle one hundred years ago and ever since by many others; Sir Andrew Clark<sup>1</sup> coined the term

<sup>1</sup> Fibroid Diseases of the Lung, London, 1906.

and made a thorough study of the pathology and symptomatology of the disease. C. J. B. and C. T. Williams,<sup>1</sup> in their book on consumption, also give a complete description of this form of phthisis. Of the more recent writers who treat of this subject, may be mentioned Bard,<sup>2</sup> Sokolowski,<sup>3</sup> and Piéry.<sup>4</sup> While most of the authors do not agree on the various points which characterize fibroid phthisis, yet in the main they are in agreement on its differentiation from all other forms of pulmonary tuberculous disease.

**Etiology.**—Fibroid phthisis is mainly encountered in persons between forty and sixty years of age and, contrary to the statements of many authors, it may occur in younger individuals. Apparently many cases are treated for chronic bronchitis, asthma, pulmonary emphysema, etc., and only after the process has lasted for many years is the character of the affection recognized; an intercurrent hemorrhage or tubercle bacilli in the sputum reveals the true nature of the disease. I have met with many cases in persons under thirty years of age.

It appears that syphilis is an important etiological factor; when both tuberculosis and syphilis are met with in the same individual, the process of the former is often of the fibroid type. Sergent<sup>5</sup> and several other French writers have indeed maintained that most fibroid cases are a manifestation of syphilis and tuberculosis. Several English authors hold the same view. Thus, J. Mitchell Bruce<sup>6</sup> says: "It should be noted that some cases of quiescent phthisis give a history of syphilis which may account for the disposition to fibrosis, and *pro tanto* may be a favorable element prognostically." In my experience, this holds true for some cases but not for the majority. I have seen many cases of fibroid phthisis in which specific disease was positively excluded, and at the Montefiore Home, where we have many of these cases, the Wassermann reaction is only rarely positive and the other stigmata of syphilis are lacking in the majority of cases of fibroid phthisis.

English authors, notably Clark, have observed that the gouty diathesis which is antagonistic to tuberculosis is responsible for the fibroid form of phthisis. This is not in agreement with my experience, because among the poor in New York City gout is rather rare, while fibroid phthisis is quite common. Nor have I found any etiological relations between fibroid phthisis and alcoholism, or social and economic conditions, etc.

It appears to me that occupation is of greater etiological moment. Most of the cases I have seen were in persons working indoors, inhaling animal and vegetable dust—garment-workers, furriers, rag-pickers,

<sup>1</sup> Pulmonary Consumption, London, 1887.

<sup>2</sup> Forms cliniques de la tuberculose pulmonaire, classification et description sommaire Genève, 1901.

<sup>3</sup> Klinik der Brustkrankheiten, Berlin, 1906, ii, 410.

<sup>4</sup> La Tuberculose pulmonaire, Paris, 1910.

<sup>5</sup> Presse Médicale, 1908, xvi, 657.

<sup>6</sup> Lancet, 1913, i, 591.

etc. It seems also that chronic lead poisoning is a predisposing factor, because of its frequency among plumbers, printers and house painters. In former days it was frequently seen among chimney sweeps, and today it is met with among those who inhale any irritative dust, as knife-grinders, coal-heavers, button-makers, etc.

**Pathology.**—The pathology of fibroid phthisis has been thoroughly studied by Sir Andrew Clark, who described that the affected lung is usually decreased in size, sometimes its dimensions do not exceed the size of a closed fist. In local fibrosis only the affected part of the lung may be contracted while the rest fills up its place by compensatory emphysema. Cavities—pulmonary and bronchiectatic—are common, surrounded by dense, rigid walls. Cheesy nodules encapsulated by fibroid tissue are frequent, and during the final stages the caseating process gains the upper hand and breaks through the limiting and protective fibrous tissue spreading the destructive process. The walls of the alveoli are thickened and finally obliterated or filled in, the interlobar connective tissue, especially around the large vessels and bronchi, proliferates enormously and, replacing the parenchymatous tissue of the lung, produces a state of induration through which the dilated bronchi pass.

In all cases of fibroid phthisis the pleura is thickened over the affected area, sometimes attaining a thickness of one-half to three-fourths of an inch. The pleural cavity is adherent and, in the pleural form, obliterated by tough fibrous tissue binding the two surfaces together, and from it other bands of connective tissue are sent forth into the lung which contract and drag along toward the affected side the mediastinum, the diaphragm, and with it the liver, etc.

We are not clear why the tubercle bacilli produce caseation and liquefaction of tissue in most cases, while in others a proliferation of connective tissue is the dominant feature after infection. We know that in many cases of fibroid phthisis we have an additional etiological factor, the inhalation of mineral, animal, and vegetable dust. But on the other hand, the form which will be described as the pleural form of fibroid phthisis is not usually associated with the inhalation of irritating dust, but the causative factor seems to be bacterial, plus the predisposing factors which are operative in the other forms of chronic phthisis.

We are in the dark about these problems. It has not been proven that in fibroid phthisis the tubercle bacilli are of some attenuated strain, or of the bovine type. In many cases of fibroid phthisis in which tubercle bacilli are not detected, Much's granules have been found, thus pointing to bacilli which have lost their acid-fast properties, being the cause; but this also requires further study.

**Forms of Fibroid Phthisis.**—The symptomatology of fibroid phthisis depends on the form of the disease. My experience is in agreement with that of Sokolowski, excepting that I meet with a pleural form in addition to his two forms—simple fibroid phthisis and fibroid phthisis

with emphysema. The most common clinical form encountered by me is the emphysematous.

**The Emphysematous Form.**—The patient has usually been a chronic cougher, expectorated for years and felt short-winded, especially on exertion, as climbing stairs. He may have consulted physicians repeatedly and was informed that the trouble was not of serious import; that it was chronic bronchitis, pulmonary emphysema, etc. Inasmuch as he has been able to pursue his occupation, he more or less disregarded the cough, expectoration, dyspnea, etc. During the winter and autumn these patients are usually subject to “colds,” “grippe,” etc., when the cough is aggravated and persists for several weeks with greater severity than usual.

In some patients, especially those engaged in trades involving the inhalation of animal or vegetable dust, the signs of pulmonary emphysema, as well as attacks simulating essential asthma are apt to come on suddenly in one who has never before suffered from any respiratory trouble. In fact, experience has taught me to look with grave suspicion on each case of emphysema or asthma coming on suddenly in a person over thirty years of age.

During the early stages of the disease, and this may last for many years, the patient, though coughing and suffering from mild dyspnea, pursues his vocation without interruption. Fever is lacking, excepting during an acute exacerbation or some intercurrent affection. The expectoration is scanty; in fact the cough is usually dry, or some glairy mucus is brought up after a fit of coughing. A search for tubercle bacilli is usually fruitless. But the dyspnea is annoying and increases on slight exertion.

The general appearance of the patient is that of a healthy person, the panniculus adiposus is well preserved, and in those who do not work at hard manual labor and in women, we may meet with marked obesity. The “fat phthisis” of which we spoke above is seen almost exclusively in fibroid patients. On the other hand, there are some patients who are more or less emaciated, but they are usually individuals who have never been fat; but even they gain rapidly after the physician urges them to rest and feed up. I have met with some who have gained twenty or even more pounds in a couple of months and retained it for years.

The vast majority of fibroid patients have clubbed fingers and curved nails. The most exquisite forms of drumstick fingers may be found among them, while they are not so common among those who suffer from common chronic phthisis.

Many get along fairly well for years without suspecting the real nature of their trouble, until they are suddenly seized by attacks of hemoptysis which may be slight, or quite profuse, but which usually frighten them out of their wits. In some, the hemoptysis is quite frequent and may at times be copious, while in most it is rare and consists only in one or two mouthfuls of blood or streaky sputum.

It may appear suddenly while the patient has considered himself in excellent condition. It may recur at irregular intervals. Hemorrhagic phthisis usually is fibroid phthisis and most patients bear the bleeding very well indeed. I had one patient who was so used to hemoptysis that it no longer frightened him. We meet with some who never expectorated blood.

Well-to-do patients without profuse hemoptysis get along for years without troubling themselves about the cause of their mild cough and dyspnea unless they apply for life insurance, and after they are rejected for "lung trouble" they promptly consult a physician.

**Physical Signs.**—A physical exploration of the chest usually reveals an emphysematous or barrel-shaped chest in those who suffered for years, while in those who have only recently acquired the disease, the thorax may be of normal shape. Careful inspection shows some flattening of the supraclavicular, infraclavicular and supraspinous fossæ, more marked on one side of the chest; wasted muscles of the neck and shoulder, and shoulder droop on the same side, coupled with lagging and restricted motion. On percussion, defective resonance, or even dulness is elicited on one side above the second or third rib anteriorly and posteriorly, while below, and all over the opposite side of the chest the note is hyperresonant or slightly tympanitic, and the inferior margin of the lung is one or two inches lower than normal and hardly mobile. Narrowing of Krönig's resonant area can easily be made out; in fact it appears somewhat exaggerated because the opposite unaffected apex is larger, owing to emphysema. Auscultation shows feeble breathing all over the chest, while over the site of the dulness the expiratory murmur is harsh and prolonged, at times showing a bronchial timbre. Dry crackles or rales after cough may be audible, in others sibilant or sonorous rales are heard all over one side of the chest. During one of the asthmatic attacks, which in some patients are quite frequent, so that they are treated for asthma, we hear wheezing, sibilant and sonorous rales all over the chest, exquisitely simulating bronchial asthma.

**Course of the Disease.**—These patients get along quite well till they pass middle age. Most of them, if they are under medical care at all, are considered individuals who are troubled with chronic bronchitis, pulmonary emphysema, asthma, etc. But sometimes between the age of forty and sixty, though exceptionally I have seen it in younger individuals, the clinical picture changes. They begin to lose weight gradually but persistently, so that sooner or later they present the unmistakable appearance of the average consumptive in the advanced stages of the disease. The cough becomes more severe and productive of globular and nummular sputum containing tubercle bacilli and elastic tissue, etc. The cyanosis and the dyspnea become more and more marked, and finally orthopnea sets in with signs and symptoms of dilatation of the right heart which is almost constant at this stage, followed by edema of the lower extremities, hydrothorax,

etc. Intestinal and laryngeal tuberculosis are quite common, and contribute to the misery of the patients who finally expire from asystole or inanition.

The signs in the chest do not differ markedly from those met with in the usual case of far advanced phthisis—signs of cavitation at the apices as well as of diffuse bronchitis are common. Skiagraphy, which in previous stages showed only signs of emphysema with some retraction of one or both apices, now reveals more or less extensive cavities and peribronchial infiltration. Displacements of the mediastinum are more frequent than in common chronic phthisis.

**Diagnosis.**—In the later stages of the disease the diagnosis is clear and it differs from that of chronic phthisis mainly because of the dyspnea, cyanosis, edema and clubbed fingers, which are not as common or less marked in the latter condition. In the earlier stages, however, fibroid phthisis is difficult to differentiate from pulmonary emphysema, chronic bronchitis and at times from bronchial asthma. The persistently negative sputum is especially perplexing. Errors may, however, be reduced to a minimum by carefully examining the apices in each case of chronic bronchitis and pulmonary emphysema. Whenever the physical signs point to infiltration of an apex, fibroid phthisis is to be thought of. The symptoms and signs of asthma coming on suddenly in one who works in surroundings laden with animal, vegetable, or mineral dust, usually point to fibroid phthisis.

**Simple Fibrosis.**—These are cases of fibroid phthisis in which the onset, course and termination of the disease are practically the same as in the form just described, excepting that the symptoms of pulmonary emphysema are lacking. The onset is slow and insidious. The patient is troubled with an occasional morning cough, expectorates little or nothing, and the sputum contains no tubercle bacilli or elastic tissue. There is, however, slight dyspnea on exertion which is often overlooked.

The general condition of the patient leaves little or nothing to be desired. He has no fever, no night sweats, no anorexia, emaciation, etc. All he complains of, if at all, is that he is subject to "colds," especially during the winter months; that he is short-winded, and of hemoptysis, which may be quite a feature in this form of phthisis when occurring often, or is copious. But before, during, and immediately after the hemoptysis there is usually no fever, and convalescence is rapid. In fact many of the patients feel much relieved after the effects of a brisk pulmonary hemorrhage have passed away. These are the cases which some English authors have described as "arthritic" or "gouty" hemoptysis (see p. 195), because some of these patients, though not all, present some of the stigmata of the arthritic diathesis.

Many of these patients present themselves to their physician who makes a careless examination of the chest and, finding no sign of tuberculous infiltration, assures them that the bleeding came from a ruptured bloodvessel in the throat, etc. Thus reassured, they return to work,

feeling quite well. However, in many there are signs of active phthisis in one of the apices: Impaired resonance, contraction of Krönig's resonant area, harsh bronchovesicular or distinctly bronchial breath sounds, more or less numerous rales, all localized, circumscribed and persistent above the second rib anteriorly and posteriorly over the supraspinous fossa in one side of the chest. The physician is often amazed to find the patient in such excellent condition for years despite the signs of a distinct and active pulmonary lesion, and is apt to attribute it to chronic apical catarrh.

In other cases the onset is, however, not so insidious. A fairly healthy person is suddenly seized with a pulmonary hemorrhage which may be slight, moderate or, rarely, copious; or he may develop mild fever, nightsweats, cough and expectorate sputum containing tubercle bacilli. A physical exploration of the chest shows a typical lesion of moderate extent. Inasmuch as for several weeks the patient presents most of the symptoms and signs of progressive phthisis, even hectic fever, nightsweats, emaciation, etc., a grave or doubtful prognosis is rendered.

But slowly the condition of the patient begins to improve; the fever abates, the cough is ameliorated or ceases altogether, the appetite improves and the patient gains in weight considerably, so that in a few months his weight exceeds that found before the onset of the disease. He considers himself cured. But a physical examination of his chest shows distinct and unmistakable signs of a smouldering tuberculous lesion in one apex; in fact all the signs of active disease are there. Feeling well, the patient reënters his occupation and works quite efficiently, believing that the physician who declared him still actively tuberculous is an alarmist. I have had patients of this class who have been doing well for years and came around to the office to "prove" it to me. Many are of the class discharged from sanatoriums as improved or even "unimproved," and inquiry in later years shows that a large proportion remain in good condition and working, except for more or less pronounced dyspnea which annoys them.

After some years the symptoms are gradually aggravated, they complain they have "caught a new cold," which is difficult to cure. The cough is persistent and exhausting, the dyspnea distressing, and they begin to lose in weight and strength progressively, presenting clearly the characteristic clinical picture of chronic phthisis with its usual complications, plus dilatation of the right heart, dyspnea and orthopnea. Physical exploration of the chest shows the usual clinical picture of cavitary phthisis, but there is in addition bronchitis, which is unusual in chronic phthisis. It differs, however, from chronic phthisis by the fact that fever is lacking or at most some insignificant elevation of temperature is noted at times. No nightsweats are present, or only slight, at the end of the disease.

**Pleural Form of Fibroid Phthisis.**—In the pleural form of fibroid phthisis, which has been graphically described by Williams, the

patient usually gives a history of an attack of pleurisy with effusion, from which he has recovered after a longer or shorter illness, the fluid having been absorbed spontaneously or was aspirated. But ever since he has remained with a dry, hacking cough, productive of little or no sputum, and in spite of the great care he has been taking of himself, he has not succeeded in recuperating completely. Dyspnea is marked and increasing steadily in intensity. In many cases the cyanosis of the fingers and face is very pronounced.

During recent years I have met with some cases of this type following artificial pneumothorax. A pleural effusion was slow in disappearing and the gas inflations had to be discontinued. But the patient kept well on the road to recovery, remaining with a pleuropulmonary tuberculous lesion.

Examination shows distinct immobility of the lower half of the side of the chest in which the effusion had taken place some retraction of the chest wall and scoliosis, or kyphoscoliosis. Mensuration shows that the affected side has fallen in—the circumference being smaller than the unaffected side by more than one inch. Vocal fremitus is absent over that area. On percussion we find dullness, at times even flatness not unlike that over pleural effusion, which is at once suspected. This is apparently confirmed by the absence of the vocal fremitus and of any breath sounds, while in some we hear distinct tubular or even cavernous breathing. There may be no adventitious sounds, but occasionally some medium-sized or large, moist and consonating rales and gurgles are audible during both phases of respiration. At times, distinct friction sounds, grating and grunts are heard.

On the unaffected side signs of pulmonary emphysema are found—hyperresonance and the inferior margin of the lung extends two to four inches lower than on the opposite side owing to emphysema, and the pulmonary retraction and upward displacement of the diaphragm on the affected side accentuates it. Anteriorly, the border of the unaffected lung extends well over the sternum.

The heart is almost invariably dislocated toward the affected side which serves as a good sign of differentiation from pleural effusion with which it may be confounded, because in effusions the dislocation is invariably toward the unaffected side, if at all. In left-sided lesions we may find the apex as far out as the axillary line and one or two interspaces higher than the normal; in right-sided lesions the apex may be found at the xyphoid cartilage, or even farther to the right. It is in these forms of phthisis that acquired dextrocardia is at times found. It is due to traction of the heart by fibrous bands in the right pleura and lung and also to the pressure exerted by the vicariously emphysematous left lung. The shrinkage, as well as the fibrous bands in the lungs also drag the diaphragm upward and, when the right side is affected, the liver is also elevated. In the left side the stomach may be elevated along with the diaphragm. Pulmonary retraction in the left side also exposes the heart and brings it near the chest walls



where we may see it pulsating. These conditions may be made out by careful percussion, but in many cases the aid of skiagraphy is necessary to clear up mooted points.

There are other clinical peculiarities which should be mentioned. Fever is usually absent throughout the course, excepting when due to some intercurrent affection. When we find a persistent elevation of temperature we may look for some complication, especially an infiltration of the opposite, hitherto unaffected lung. The cough, which was moderate for a long time, in some cases for years, becomes more and more severe and the amount of sputum brought up may be enormous. Both the cough and the expectoration may be influenced by posture—the patient coughs more when lying on one side and somewhat relieved when turning on the other side, just as in bronchiectasis. This, however, gives no clue as to which side is affected. The sputum contains tubercle bacilli in large numbers and is at times fetid, which is rare in other forms of phthisis.

Hemoptysis, which is very frequent in other forms of fibroid phthisis, is less often encountered in the pleural form. But when occurring, it is apt to last for days or weeks and at times it is copious. I have seen two cases in which it was the cause of death of patients who were otherwise getting along very well.

The dyspnea, which is a feature of all forms of fibroid phthisis is more severe in this type because of the loss of lung tissue and the displacement of the heart. In fact I have seen many cases in which the lesion in the lung was practically healed, or at least distinctly inactive, yet the dyspnea was severe and even unbearable. Another feature is cardiac palpitation, especially in left-sided lesions, which is apt to be so severe as to make life unbearable.

In the terminal stages signs of cardiac dilatation set in, edema of the lower extremities, enlargement of the liver, cyanosis, etc., and the patient dies from asystole. In many cases complications are responsible for the final outcome—hemorrhage, which was already mentioned, inanition due to laryngeal tuberculosis with dysphagia, amyloid degeneration of the various visceral organs, etc. Tuberculosis of the previously unaffected lung may bring about a rapid course of the disease.

I have observed that some of these cases, tuberculous in origin as they are, become purely bronchiectatic. The tubercle bacilli disappear from the sputum, but the patient continues to cough and expectorate large quantities of sputum which shows all the characteristics of sputum in bronchiectasis; in fact, the course is that of non-specific bronchiectasis after this occurrence.

**Prognosis in Fibroid Phthisis.**—As regards duration of life, fibroid phthisis, though an active tuberculous disease and hardly ever cured, is more favorable than the other forms of phthisis excepting abortive tuberculosis. It is among the fibroid patients that we find individuals who have been tuberculous for years. I have some who have lasted

for twenty-five years, and Sokolowski reports one who lasted for more than forty years. While they are always ailing, many are still fit to pursue their vocation, and I have among my clientele some who have worked quite hard without long interruptions.

In fibroid phthisis, the reparative processes of nature are more active than the destructive tuberculous, and the patients are shielded from the extension of the caseating and softening processes, the fibrous tissue usually forming a wall around the lesion limiting its progress and preventing the absorption of toxins, as is evident from the absence of fever, etc. Because of the pleural adhesions the patients are shielded from such complications as spontaneous pneumothorax, which never occurs among them. When in my hospital practice I once found a fibroid patient presenting the symptoms, of spontaneous pneumothorax, it was soon clear that the rupture occurred in the lung which had been unaffected, but recently showed a new lesion.

## CHAPTER XXIII.

### ABORTIVE TUBERCULOSIS.

**Natural Resistance Against Phthisis.**—As was already shown, infection with tubercle bacilli is harmless to the vast majority of civilized people; the lesion cicatrizes more or less quickly without producing distinct clinical symptoms. During childhood, when most infections occur, the morbidity and mortality from this disease are insignificant.

We cannot recognize these mild or abortive infections clinically, except by the tuberculin test; they probably pass as slight or severe "colds," grippe, bronchitis, etc. Nor do we know whether they are due to the inoculation by strains of bacilli of low virulence, considering the marked difference in virulence displayed by various strains of tubercle bacilli. The suggestion that they may be due to infection with bovine bacilli appears to have much in its favor, but this also has not been proven.

We meet at times cases of abortive tuberculosis, *i. e.*, patients in whom the disease, instead of pursuing the usual clinical course to its termination in death or recovery after several months' or years' illness, is aborted within a few weeks or months of indisposition. In other words, just as we at times meet with cases of abortive pneumonia, typhoid, scarlet fever, etc., so is there a form of pulmonary tuberculosis which is of relatively short duration and invariably terminates in recovery. In these cases the lesion is apparently circumscribed, of little activity, often altogether latent and quickly cicatrizes, and when the patient dies from any other cause it is found at the autopsy in the shape of more or less extensive scars located at the extreme apex, pleural adhesions, or even isolated fibrous or calcareous nodules which hardly caused any inconvenience to their owners during life.

In the older works on phthisis, this form of tuberculosis is not mentioned at all. In former days only advanced phthisis was recognized. But in recent years, since Bard<sup>1</sup> described the pathology and symptomatology of *tuberculose abortive*, many others have mentioned it more or less extensively. In the second edition of Cornet's<sup>2</sup> treatise, also in Bandelier and Röpke's book, as well as in Minor's article in Klebs' treatise, we find it mentioned cursorily, while Piéry<sup>3</sup> in his book devotes an extensive chapter to it. Bezançon<sup>4</sup> and the present author<sup>5</sup> have published papers on the subject of abortive tuberculosis.

Abortive tuberculosis is responsible for a large proportion of "non-tuberculous" cases in sanatoriums—the lesion heals very quickly and

<sup>1</sup> Formes cliniques de la tuberculose pulmonaire, Genève, 1901.

<sup>2</sup> Die Tuberkulose, Vienna, 1907, p. 690.

<sup>3</sup> La tuberculose pulmonaire, Paris, 1910, p. 491.

<sup>4</sup> Bull. Soc. intern. hôp. de Paris, 1901, p. 933.

<sup>5</sup> Medical Record, 1913, lxxxii, 921.

it is often suspected that the patients were admitted through an error in diagnosis. Many of the patients who state that well-known physicians have considered them tuberculous at one time, but that they have none the less been healthy all along for years, have in fact been affected with the abortive type of the disease at the time the diagnosis was made. I have seen many patients who applied for admission to public sanatoriums and were passed by the admitting physicians as eligible incipient cases, but inasmuch as the institutions were overcrowded, they had to wait for weeks or months for vacant beds. When they were finally called, it was found that all the symptoms and signs of the disease had vanished. A large proportion of cases of "persistent colds," grippe, rhinopharyngitis, etc., are also abortive tuberculosis. If they were carefully studied, we would discover some physical signs in the chest substantiating this view. In fact, L. Napoleon Boston<sup>1</sup> reports finding tubercle bacilli in cases of acute colds, influenza, bronchitis, etc., but the patients recovered without becoming tuberculous. Many of these were in fact abortive tuberculosis.

**Symptomatology of Abortive Tuberculosis.**—The symptoms and signs of abortive tuberculosis are the same as those of incipient phthisis, but they never pass beyond that stage. In most cases it begins with the symptoms of a common "cold." After some exposure the patient begins to cough, has some fever, malaise, backache, etc., and is treated for coryza, grippe, tonsillitis, etc. But instead of ameliorating within a few days or a week, the symptoms persist for a month or two. In many cases the onset is marked by hemoptysis. The patient, who has felt quite well, or at most has coughed for a few days, suddenly feels some irritation in the throat and coughs out some blood or blood-streaked sputum. The bleeding may last for a few hours or days and either stops abruptly or continues for a few days in the form of streaky sputum. Every physician has among his clientele patients who have expectorated blood years ago, but have felt well all along. While in many of these, the hemorrhage was of extrapulmonary origin, as was already shown, in others it was due to abortive tuberculosis.

When the thermometer is carefully and judiciously used, we find fever of a mild type; especially in the afternoon there is a rise of one or two degrees and in the early morning there may be some subnormal temperature. In some cases that came under my observation I found the typical temperature curve of mild incipient phthisis, and there were many of the accompanying symptoms of hyperthermia—malaise, languor, pain in limbs, backache, etc. While the patient is not completely incapacitated, yet he feels tired during the afternoon, but recuperates in the evening or feels refreshed after a night's sleep. Night sweats are rare, but in a few I have noted that they were drenching. The appetite is usually retained, and when the patient is told to eat well and plenty, he finds no difficulty in following instructions.

Cough is a constant symptom; though many state that they do

<sup>1</sup> Interstate Med. Jour., 1914, xxi, 330.

not cough, careful inquiry reveals that they clear their throat in the morning. We often meet with dry, hacking cough which is an annoyance during the day and keeps the patient awake during the night. Occasionally the cough is productive of glairy mucus, but the mucopurulent sputum of phthisis is never seen in abortive cases, unless there is some rhinopharyngitis.

Most abortive cases are of the "closed" variety of tuberculosis, but now and then we meet with one showing tubercle bacilli in the sputum. The albumin reaction of the sputum is almost invariably positive in these cases, and I consider it of diagnostic importance. Edward G. Glover<sup>1</sup> found that the complement-fixation test for tuberculosis is of value in the determination of the nature of some of the dubious cases.

In some, we meet with hoarseness lasting intermittently for a few hours during the day, or for several days in succession.

Tachycardia is not a very frequent symptom, but we very often find instability of the pulse; the least exertion or excitement raises its rate to 90 or more per minute. The blood-pressure is usually lower than normal. With the improvement in the condition of the patient both the pulse and the blood-pressure become normal again.

**Physical Signs.**—The objective signs are those of incipient phthisis. Of course, when the lesion is limited and centrally located, we may not find any physical signs at all and without hemoptysis and tubercle bacilli in the sputum, the diagnosis cannot be made. In all probabilities the vast majority of tuberculous infections in man are of this character. They are aborted without revealing themselves in any way. But in those in whom the conglomeration of tubercles is large enough to alter the air content in a limited area of the lung, we may find signs on percussion and auscultation.

A short note above and immediately beneath the clavicle is quite common. But this may be obscured by vicarious emphysema, hyperfunction or relaxation of the surrounding lung tissue which may emit a hyperresonant note. Shortening of an apex, or narrowing of Krönig's resonant areas is more common and can be easily made out with careful percussion.

On auscultation we may hear feeble breath sounds over the site of the lesion, or rough, interrupted, cog-wheel breathing. Only the inspiratory murmur is usually altered, but I have seen cases in which the expiratory murmur was prolonged, and even bronchovesicular in character, indicating extensive infiltration, yet recovery went on speedily, showing that even a considerable focus may be aborted. This is confirmed by the large scars, or encapsulated and calcified tubercles found at times while making autopsies on persons who died from causes other than tuberculosis.

Adventitious sounds are not often heard excepting in those who have had hemoptysis and in some grippal cases, in which dry crackles or crepitation may be audible during inspiration and influenced by

<sup>1</sup> Quarterly Journal of Medicine, 1915, viii, 339.

cough. Of course, to be of significance, these signs must be strictly localized at one apex, and constant for some time. They must also be differentiated from spurious rales, as well as from marginal sounds.

Skiagraphy is of little value as was already stated in Chapter XVII.

**Diagnosis.**—These are the classical symptoms and signs of incipient phthisis, and when meeting with a case we are by no means certain as to the course the disease is likely to take. In fact, many abortive cases are admitted to sanatoriums where they are speedily cured, and they contribute no small portion of the statistical success of institutional treatment.

In the progressive cases the lesion extends and the constitutional symptoms become more and more marked within a few months, while in the abortive forms the mild fever, cough, nightsweats, etc., abate within a few weeks or one or two months, and the physical signs disappear, or they are superceded by sibilation and there may permanently remain a prolonged expiratory murmur over the affected apex. While in most cases the local impairment of resonance remains, and for this reason there are many persons in whom there are differences in this regard when the two apices are compared, I have observed that in some even this disappears, to be replaced by slight hyperresonance, due probably to hyperfunction, the result of vicarious emphysema of lung tissue around the cicatrix which was caused by the healing process.

Without observing the patient for several weeks, and without an initial pulmonary hemorrhage, or tubercle bacilli in the sputum, abortive tuberculosis cannot be diagnosticated, because there always lurks a suspicion that it may have been a non-tuberculous apical lesion. There are, however, some points which may help us in recognizing this form of tuberculosis: When a patient with an apical lesion has a good appetite, and normal gastric function, gaining weight and strength as soon as he begins to take care of himself, there is a likelihood that the lesion may be aborted and cured within two or three months. However, this may prove deceptive at times. Some points which have helped me are the following: A slow pulse, not much influenced by exertion or excitement, speaks for a benign process. The initial hemoptysis of chronic phthisis, as was already stated, is usually preceded by cough, weakness, nightsweats, etc., for weeks before the bleeding, while in abortive cases this is rare—the hemoptysis comes like a thunderbolt out of a clear sky, without any premonitory symptoms and without any apparent exciting cause. In progressive cases the initial hemoptysis is usually more abundant, and always followed by fever of the type described above. In abortive tuberculosis the temperature remains normal at times, but usually it is slightly elevated,  $1^{\circ}$  or  $1.5^{\circ}$  for a couple of weeks. Initial hemoptysis of tuberculous origin without high or moderate fever, and without tachycardia, weakness, languor, etc., points to an abortive lesion.

In the majority of cases, however, only careful observation of the course of the affection is decisive.

## CHAPTER XXIV.

### PULMONARY TUBERCULOSIS IN CHILDREN.

**General Characteristics of Tuberculosis in Children.**—In children infection with tubercle bacilli, if it causes active disease at all, is usually followed by a generalized morbid process with implication of the lymphatic glands. This characteristic is the more accentuated the younger the child. In fact, in all infectious diseases we may note that the reaction of the lymphatic glands is intense in children. The glands are particularly sensitive to tuberculosis.

The localized disease of the lungs peculiar to phthisis in adults, or in the bones and joints, characteristic of early childhood, is never seen in infants. "In children who have passed the seventh or eighth year the pathological process resembles that seen in adults," says Holt,<sup>1</sup> "but in younger children, and especially in infants, nothing corresponding to it is met with." In infants tuberculosis is an acute, general infection, like typhoid or septicemia, and when the bacilli localize themselves by metastasis in any part, they produce lesions akin to those of pyemia.

Because of the implication of the glandular system, especially the intrathoracic glands, it was assumed by many authors that infection in children is invariably accomplished by inhalation of the bacilli. The microorganisms are deposited in the lungs, and when attempting to invade the blood, they are retained by the lymphatic glands. When the localization of the lesion was found in the mesenteric glands, it was clear that ingestion of the bacilli was the channel of entry, and this was confirmed by the fact that in mesenteric tuberculosis bovine bacilli were often found.

But we have seen that this is not necessarily the case. Entering *via* the digestive tract, the bacilli may reach the tracheobronchial glands with as much ease as when entering *via* the respiratory tract. Behring and Calmette and their school maintain, in fact, that all tuberculosis, especially in children, is lymphogenic and hematogenic (see p. 47).

From the facts presented in the chapter on phthisiogenesis it is clear that tuberculosis during infancy and childhood is hematogenic, irrespective of the portals of entry of the bacilli. A study of the rates of mortality during the various ages of life confirms this view. As will be seen from the accompanying diagram (Fig. 69), pulmonary tuberculosis is a frequent cause of death in infants under two years

<sup>1</sup> Dis. of Infancy and Childhood, p. 1027.

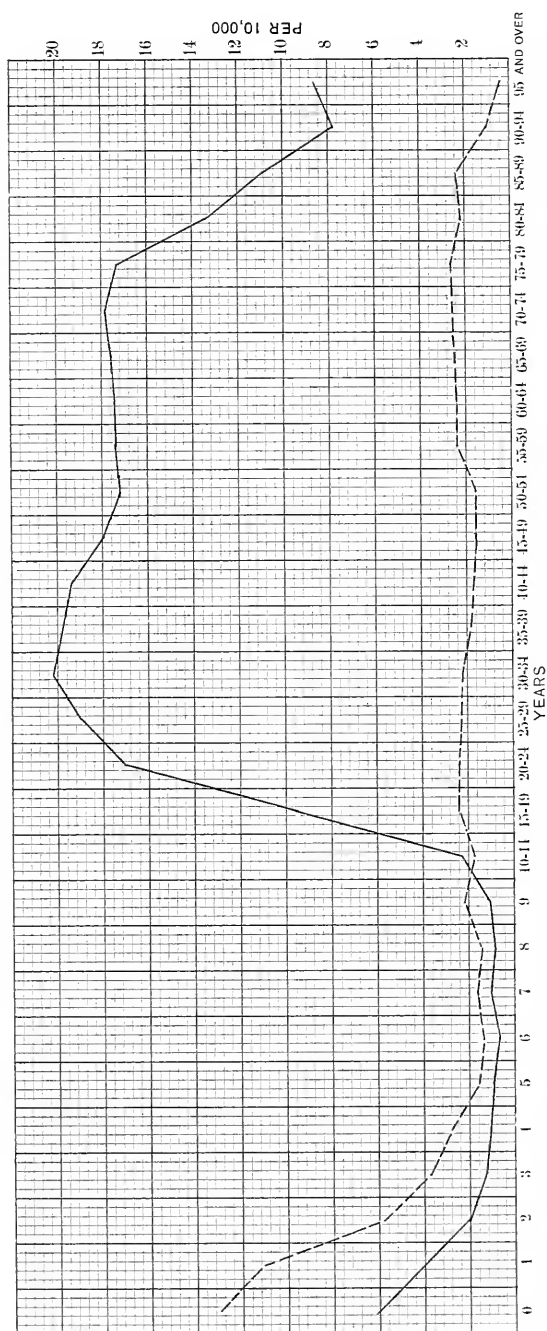


FIG. 69.—Mortality from tuberculosis per 10,000 living at the indicated age periods in the United States Registration Area, 1910 to 1913. Black line represents pulmonary tuberculosis; dotted line all other forms of tuberculosis.



of age; between three and fourteen years of age comparatively few succumb to this form of the disease, only after fifteen years of age does it become very frequent and remains so till the age groups above eighty years. We know from clinical experience that, when occurring during the first two years of life, pulmonary tuberculosis is invariably an acute disease, and the chronic type is unknown at this age. On the other hand, all other forms of tuberculosis, including that of the glands, bones, joints, serous cavities, especially the meninges, and the intestines, in short, the hematogenic forms of tuberculosis, cause death most frequently during the first four years of life, and are comparatively uncommon as a cause of death after the fifth year of life.

It is thus clear that acute tuberculosis, as well as the hematogenic forms of this infection, have a different age incidence when compared with chronic phthisis, the disease which creates the main problem. Moreover, as was already shown, during the years when most of the human infections take place, between the second and the fourteenth, the mortality from all forms of tuberculosis is comparatively low; even hematogenic tuberculosis as a cause of death maintains the same rate throughout the rest of human life. It also shows that phthisis, which is a common cause of death in adults, is not necessarily preceded by infection with tubercle bacilli immediately before the disease manifests itself by symptoms. It shifts the problem of infection from the adult to the child.

**Tuberculosis during Infancy.**—We have shown that the child is born free from tuberculosis, and that infection, if it takes place at all, occurs postpartum. Virchow, whose autopsy experience was as immense as that of any physician, stated that he never encountered a case of fetal tuberculosis. Infection in an infant is therefore invariably primary and almost always followed by symptoms of disease. Indeed, as we have already shown, there are cases on record in which infants brought into contact with a consumptive for an hour or so developed tuberculous disease of a malignant type. When the infection is massive, acute general tuberculosis with implication of the glandular system, and often of the lungs, is almost invariably caused.

The infant's organism behaves after a primary infection just as the very susceptible guinea-pig; the reason being that there is a primary infection of a body which has not yet been immunized by a previous mild infection. These cases are mostly seen in infants who live with tuberculous persons—the father, mother, sister, brother or nurse being tuberculous and, in handling the infant, an opportunity is afforded to transmit the disease. There is evidence tending to show that in some cases, though in less than is generally supposed, the infection is derived from bovine bacilli through milk from tuberculous cows.

In many cases no exciting cause, except the source of infection, can be traced. In others some acute endemic disease of infancy is found to have produced a state of allergy. This is especially true of

measles and whooping-cough, but any of the other contagious diseases of infancy may reduce the vitality and resisting powers of the infant and infection is then followed by the characteristic acute form of tuberculosis.

**Symptoms.**—The symptoms depend on the mode of onset and on the parts of the body which bear the brunt of the infection. In those in whom tuberculosis follows in the wake of another disease, like

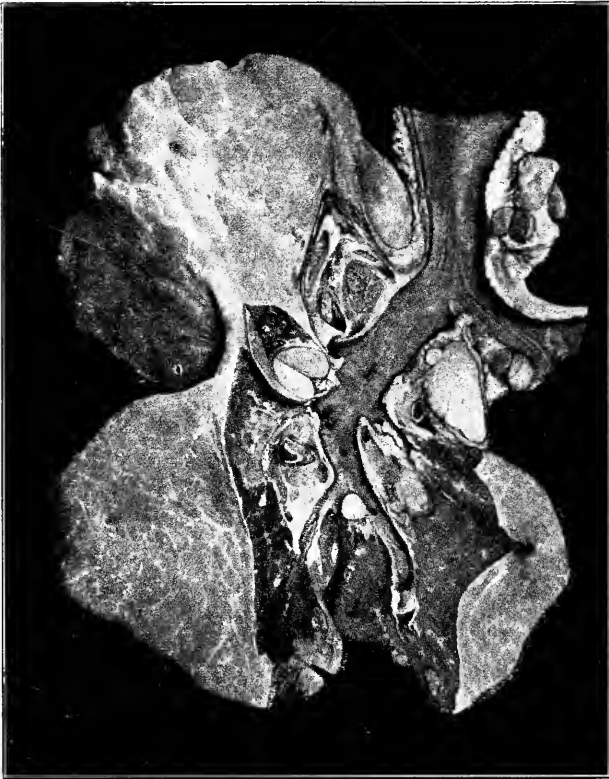


FIG. 70.—A primary cheesy focus the size of a lentil in a bronchus of the left lower lobe with miliary and conglomerate tubercles of the regional peripheral atelectatic lung. Caseation of the bronchopulmonary and lower tracheobronchial glands in the region of the right lower lobe. The glands on the left side are free. (Anton Ghon.)

whooping cough, measles, etc., there are usually symptoms of bronchopneumonia or meningitis, which carry off the patient within a few days, a week or two. In addition to the symptoms and signs of bronchopneumonia, there is often found enlargement of the spleen and liver and swelling of the superficial glands, the cervical, axillary, inguinal, etc. This form of acute tuberculosis is best seen in cases of tuberculous disease engendered by inoculation, as in infection of the wound

after ritual circumcision. Arluck and Wincouroff,<sup>1</sup> and Holt<sup>2</sup> have recently described such cases in detail.

In those in whom the disease is slower in development, athrepsia is seen. It is noted that the child does not thrive despite the fact that its nourishment leaves little or nothing to be desired and the gastrointestinal functions are fairly normal. There may be no fever at all. Still the emaciation proceeds frightfully. In some cases the emaciation consumes nearly all the subcutaneous adipose tissue and the thin, pale skin is stretched over the atrophied bones. These infants usually have long hair on the back between the shoulder blades and on the extremities; their eyes are sunken and the eyelashes are unusually long. Finally the temperature begins to rise and may reach very high, and they succumb to symptoms of septicemia or meningitis.

Examination of the chest may not show any changes, while in some we may find areas of defective resonance, bronchial breathing or rales. In infants limited and circumscribed lesions are very difficult of localization because we have no assistance on their part while exploring the chest.

Cough may be absent altogether, but in some cases we meet with a peculiar cough caused by pressure of enlarged glands on the bronchi, or on the nerves passing through the chest. Eustace Smith<sup>3</sup> first described this cough as spasmodic, occurring irregularly in paroxysms like those of pertussis, lasting only a short time and ending sometimes, though rarely, in a crowing inspiration. This cough has since been differently described by various authors. Schick<sup>4</sup> describes a respiratory crow or stridor resembling the sound heard in asthma and in capillary bronchitis. It can, however, be distinguished from the latter by the fact that in asthma the cough is paroxysmal while the stridor in bronchial adenopathy in infancy is continuous, lasting without change for weeks and months. The French have described it as *toux coqueluchoide*, and Stricker compares it with the bark of a hoarse puppy.

In most of these slow cases the cachexia progresses till finally the child succumbs to some intercurrent disease or to tuberculous bronchopneumonia. On rare occasions a softened gland ruptures into a bronchus causing aspiration pneumonia. A relatively large proportion end up with tuberculous meningitis. Investigations made by the writer<sup>5</sup> in children under six years of age living a tuberculous milieu in New York City have shown that 16 per cent. succumb to meningitis, as against only 2.6 per cent. among the general population.

Other infants may be anemic and underfed for months. They do

<sup>1</sup> Beitr. z. klin. d. Tuberkulose, 1912, xxii, 341.

<sup>2</sup> Jour. Amer. Med. Assn., 1913, lxi, 99.

<sup>3</sup> Wasting Diseases of Infants and Children, London, 1878.

<sup>4</sup> Verhandl. d. Ges. f. Kinderheilkunde, xxvi, 1909, 121.

<sup>5</sup> Archives of Pediatrics, 1914, xxxi, 197.

not thrive in spite of all efforts to improve their nutrition. Finally, the marasmus assumes an acute character, the fever rises and they succumb to exhaustion or more commonly to some intercurrent disease.

**Diagnosis.**—It is clear that the diagnosis of tuberculosis in infancy is not an easy matter. Hamburger's<sup>1</sup> advice should be followed by all who have infants under their care: *Think of tuberculosis in every case in which no other diagnosis can be made.* This dictum is shared by nearly all other pediatricists who have given thought to the problem. Tubercle bacilli cannot be discovered because infants do not expectorate. Holt has, however, often found them by swabbing the throat with a pledget of cotton. A positive tuberculin (von Pirquet) reaction in an infant under one year is sufficient to clinch the diagnosis. Unfortunately during the course of measles, or whooping-cough and in tuberculous meningitis, the tuberculin reaction is apt to be negative, despite the presence of tuberculous infection.

**Prognosis.**—The prognosis of tuberculosis in infancy is very gloomy. In fact it may be stated that the younger the infant the more unfavorable the prognosis. During the first three months of life hardly any survive infection; the vast majority of those infected during the second three months of life succumb to the disease or to some intercurrent infection; the outlook for infants between six and eighteen months is very unfavorable when infected with tuberculosis.

In this gloomy prognosis nearly all authorities agree: Holt<sup>2</sup> holds that the outlook for a young child with general or pulmonary tuberculosis is always bad; Schlossmann<sup>3</sup> says that he does not know of a single case in an infant which resulted in recovery; von Pirquet maintains that 90 per cent. of infants infected during the first year of life perish; Louis Guinon<sup>4</sup> says that before the fourth year of life tuberculosis is always fatal; and Monti<sup>5</sup> says that he never saw a case of tuberculosis in an infant under two years recover.

It appears to be the consensus of opinion of most pediatricists that all tubercles during the first two or three years of life are active, that the lungs show no tendency to limitation of the disease and that there are no reparative processes to be noted when examining the lungs of children who succumbed to tuberculosis. No cicatrization or calcification is to be observed.

The corollary has been drawn that all infants showing signs of infection with tubercle bacilli—a positive von Pirquet reaction—are doomed. The writer cannot agree with this. We have followed infants showing positive von Pirquet reactions during the first three months of life growing into healthy children. It appears that the dangers of developing active tuberculous disease, and the acuteness of

<sup>1</sup> Brauer, Schröder, and Blumenfeld's *Handbuch d. Tuberkulose*, Leipzig, 1915, v, 6.

<sup>2</sup> *Dis. of Infancy and Childhood*, 5th edition, p. 1004.

<sup>3</sup> Pfandler and Schlossmann's *Diseases of Children*, Philadelphia, 1912, ii, 632.

<sup>4</sup> *La prat. des mal. des enf.*, Paris, 1911, iv, 479.

<sup>5</sup> Ueber Tuberkulose. *Kinderheilkunde in Einzeldarstellungen*, 1901.

the process engendered, are in inverse ratio to the age at which the infection takes place. The younger the infant the more unfavorable the prognosis. But even among very young infants cicatrization and calcification of the lesion may occur. In another place I have collected evidence showing that such healed lesions were found at autopsies made on infants who died from other causes.

### TUBERCULOSIS DURING EARLY CHILDHOOD.

**Significance of Tuberculosis during Childhood.**—In our study of the epidemiology of tuberculosis we have seen that the child is born free from tuberculosis but that soon after birth, on coming into contact with tuberculous individuals or their discharges, or consuming milk from tuberculous animals, it is infected with tubercle bacilli. We have also shown that during the first year of life relatively few—between 5 and 10 per cent.—are infected with tubercle bacilli. During the second year more are infected, and the number of infections keeps on growing so that at the age of fifteen over 90 per cent. show unmistakable signs of harboring tubercle bacilli in the body. A study of the mortality from tuberculosis according to age groups has shown that the mortality from this disease is very high during the first two years of life. Considering the malignant clinical forms of the disease which have been described above, the reason is clear. But beginning with the third year the number that succumbs to this disease is very small and this low mortality keeps on till the fifteenth year, when there is another increase which keeps on rising, so that from the twentieth year onward the maximum has been reached which keeps up till far-advanced age.

It is thus clear that during the years when most infections with tubercle bacilli take place, the mortality is at its lowest. It is also clear that if infection is to take place, which we have shown to be inevitable for those living in large industrial towns and coming into contact with many people, it is best that it should occur during childhood. Apparently, during this age period death due to tuberculosis is exceptional. This point will be discussed again when speaking of the prophylaxis of tuberculosis.

**Infection and Morbidity.**—We must again emphasize the difference between infection with tubercle bacilli and disease due to this micro-organism. It appears that the vast majority of children infected with tubercle bacilli do not show any clinical manifestation of disease, otherwise over 50 per cent. of children in large cities would be sick and in need of careful treatment; at the age of ten over 75 per cent. would be sick and in need of dietetic, specific, institutional or climatic treatment. Scientific tests prove conclusively that the vast majority of children have been infected, and but few show clinical manifestations of disease; hence the bulk of infections at that age cause no disease,

and may be disregarded by the clinician. Some, however, do show clinical manifestations of disease.

**Tuberculous Tracheobronchial Adenopathy.**—Excluding tuberculosis of the bones and joints and the meninges, the bulk of the tuberculous morbidity is caused by tuberculosis of the glands, especially the cervical and the intrathoracic. In most of the children having enlarged tuberculous glands the symptoms are negligible, or there are no clinical manifestations at all. Thus we often discover enlarged glands on the neck or in the thorax of children who are in an excellent condition of health. In some we find the glands enlarged for some time, then there is recession, the swelling goes down or disappears, while the children kept up their activities at school, and were none the worse for the experience. In others the appearance of the glands is concurrent with the occurrence of some disease, like measles, scarlet fever, whooping-cough, etc.; they remain enlarged during convalescence, but after complete recovery they recede or disappear permanently, or may return when some other exciting cause is again operative. We may thus see in many children a tendency to enlargement of the glands whenever an exciting cause is operative, but the innate forces of resistance are at work and recovery takes place in a short time, spontaneously or after some treatment has been instituted. This class of children needs no special treatment beyond life in healthy surroundings and good nourishment.

**Symptoms of Glandular Tuberculosis in Children.**—In others the appearance of glandular tuberculosis is accompanied, often preceded, by symptoms which are troublesome and need careful study for their recognition.

Of these symptoms the following are the most important: Emaciation, fever, night sweats, anemia, anorexia, etc.

**Emaciation.**—A healthy child gains in weight constantly, and if it is regularly weighed, say every month, it will be found that the scale registers more than at the preceding weighing. While in normal adults a lack in this direction is not necessarily an indication of disease, because they may have reached their normal standard, or even exceeded it, with children conditions are different. Commensurate with their gain in height, there must be a gain in weight in children of school age. It is known as the normal increment in the size of the body. When a child does not gain in weight it is, with few exceptions, an indication of disease.

To ascertain this gain in weight various tables have been prepared by anthropometrists and reproduced in many text-books on pediatrics. But I want to warn the practitioner against comparing the weight of a child under his care with that given in any of these tables. To begin with, the weight given in the table for each age is an average of a large number of children, and averages permit variations that are normal. The weight of the child depends solely on its height, and there are perfectly healthy children and adults who are short of stature.

What the physician should look for is a steady gain. If this is not found, it is clear that the child is sick. At any rate, it demands an explanation. In many cases it may be because of some intercurrent non-tuberculous disease. But it should be found and treated. When we find that a child is not gaining in weight for several months, it is equivalent to a steady loss in an adult. If there is no morbid condition to account for it, tuberculosis may safely be suspected as the cause. A careful physical examination will, in the majority of cases, reveal enlarged intrathoracic glands.

An exception is to be mentioned. Infants may be suffering as a result of tuberculous infection and show no signs of emaciation for a long time. This is evident from the fact that tuberculous meningitis or bronchopneumonia often attacks well-nourished infants. Infantile tuberculosis, unless the gastro-intestinal tract is affected, does not often lead to cachexia.

With the emaciation there is often to be observed anemia, manifesting itself in marked pallor of the skin and mucous membranes, though an examination of the blood may not disclose any definite changes in its cytology.

**Fever.**—Whenever tuberculous glands cause trouble there is a rise in temperature. Hamburger's conception of tuberculous disease supplies the theoretical basis for the fever in these cases. He looks upon all clinical exacerbations of tuberculosis as spontaneous tuberculin reactions due to a sudden flooding of the body juices with tuberculin, producing the same symptoms as we produce artificially by injecting tuberculin.

The healthy child's temperature oscillates between 98.8° and 99.8° F. Whenever it rises above these limits, it is to be considered pathological and an explanation is to be sought. If no cause can be found for elevation of temperature, which is observed persistently for several weeks, tuberculosis is to be thought of. In most cases it will be found that in addition to the thermometrical findings there are also symptoms of hyperthermia, such as anorexia, languor, etc. The child may feel refreshed and lively during the morning hours, but late in the afternoon it is flushed, tired, and seeks rest.

In evaluating thermometrical findings it must always be remembered that the fluctuations in the temperature are much more pronounced in children than in adults. Thus among children in Chicago, Th. Sachs<sup>1</sup> found that the morning temperature fluctuated between 98.4° and 100.4° F. and the afternoon temperature between 97.4° and 100.5° F. E. Wynne<sup>2</sup> found that among 1000 children 261 had temperatures of 99° F. or over, and of these, 112 presented no obvious pathological condition to account for the hyperthermia. Mary E. Williams<sup>3</sup> found among 1000 school children between the ages of

<sup>1</sup> Sixth Intern. Congr. on Tuberc., 1908, ii, 479.

<sup>2</sup> Public Health, 1913, xxvi, 136.

<sup>3</sup> Lancet, 1912, i, 1192.

twelve and fourteen years no less than 55.5 per cent. had temperatures of 99.6° F. and higher.

There are two reasons to account for the oscillations of the temperature in children. The heat centre is more apt to be disturbed by slight factors than in adults, as is shown by the fact that nearly all pathological conditions produce higher fever in them than in adults. Then, there are so many subacute or chronic conditions which produce mild fever in children, that it would be wrong to base a diagnosis of tuberculosis on thermometrical findings alone. But when the temperature is found elevated persistently for several weeks in a child, and other symptoms of tuberculosis are present, while no other cause can be discovered, the patient is to be kept under careful observation. A difference of more than 1.5° F. between the minimum and maximum temperature of the day, when persistent, points to tuberculosis, when no other cause can be found.

**Nightsweats.**—As a symptom of tuberculosis in children nightsweats have not the same significance as in adults. Many non-tuberculous children sweat during the night. In a study of the physiological phenomena of sleep in children, Czerny<sup>1</sup> found that the intensity of evaporation from the skin goes hand-in-hand with the depth of the sleep. At the time when sleep is most intense, at its maximum, the skin is warm and moist, and usually profuse perspiration on the face is noted. This is not to be considered pathological.

To be of diagnostic significance, nightsweats in children must appear during the second half of the night and be so profuse as to soak through the bedclothes. Even in such cases they may not be pathognomonic of tuberculosis; the possibility must always be borne in mind that they may be of nervous origin, especially in older children. At any rate, nightsweats are often absent in tracheobronchial adenopathy, though with each exacerbation of the symptoms of activity, they are to be observed.

In tuberculous bronchopneumonia in children nightsweats are the rule, but in non-tuberculous cases they are often a prominent and annoying symptom.

**Cough.**—Cough is another symptom of active tuberculosis in children. Hamburger says that it is never absent in active, incipient cases, and when a cough lasts more than a week the possibility of tuberculosis should be considered and a thorough search for other symptoms and signs of the disease should be inaugurated. In advanced stages of the disease cough may be lacking, especially when there is an arrest in the progress of the disease, which is not infrequently the case in children between eight and fourteen years of age. But even in these cases we meet with frequent exacerbations of the disease when the child coughs more or less. According to Schlossmann, Holt and others, cough may be entirely absent in infants with active disease.

<sup>1</sup> Jahrb. f. Kinderheilk., 1892, xxxiii, 22.



We must, however, emphasize that in children over three years of age cough is only of significance as a symptom of active tuberculosis when other symptoms are present, especially emaciation. When a child thrives, despite a chronic cough, it will be found that there is another cause, especially chronic or subacute catarrhs of the nose and

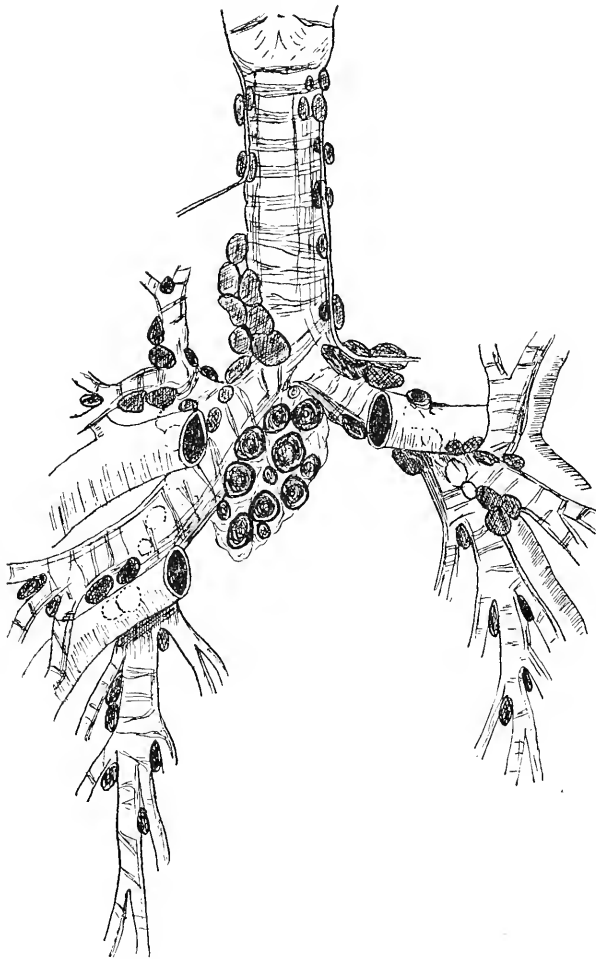


FIG. 71.—Diagram showing greater number of glands located on the right side.

throat, particularly during the winter months. Asthma also is often a cause, and so is chronic bronchitis, though we must be careful when finding unilateral bronchitis, which is almost invariably tuberculous.

The paroxysmal and the brassy cough of infants, as well as the expiratory stridor of infants have already been described.

Children presenting any or all of these symptoms—emaciation, fever,

nightsweats, cough, etc.—require a careful physical examination and if these symptoms are due to active tuberculosis, we almost invariably find local tuberculous changes—that the glands are affected—except in those over eight years of age, among whom localized pulmonary tuberculosis of the same character as in adults may be found.

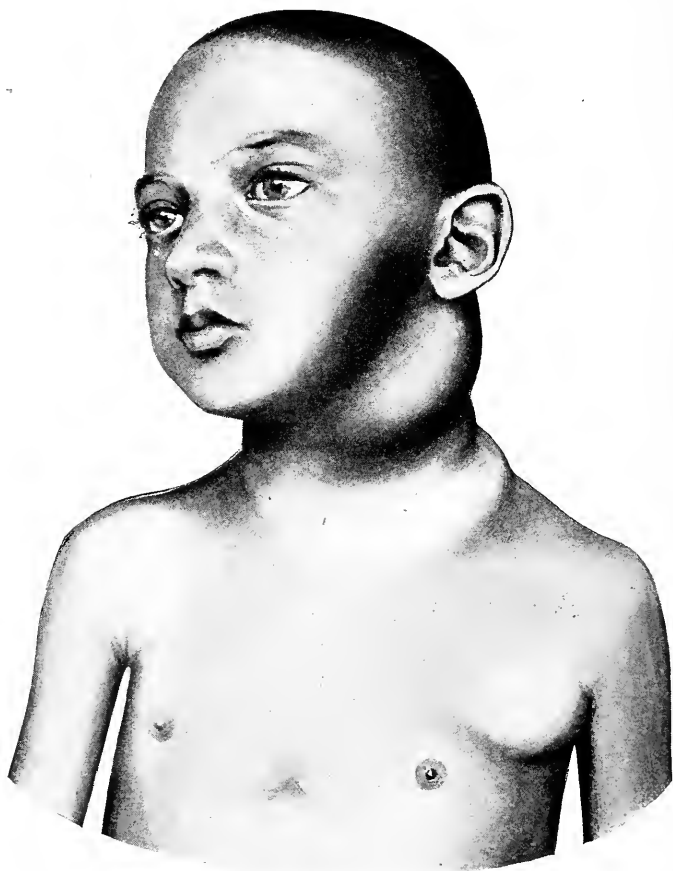


FIG. 72.—Tuberculosis of cervical and axillary lymph nodes in an eight-year-old boy.  
(Carr.)

**Cervical Adenopathy.**—Among the glands most frequently affected in active tuberculosis in childhood the most important are the cervical and the tracheobronchial. The former group is easily examined because when enlarged, we can see and palpate them and ascertain their condition.

If we should take enlarged cervical glands as an indication of active tuberculosis in children, we would find very few raised under adverse hygienic and economic conditions who are free from the disease. Thus,

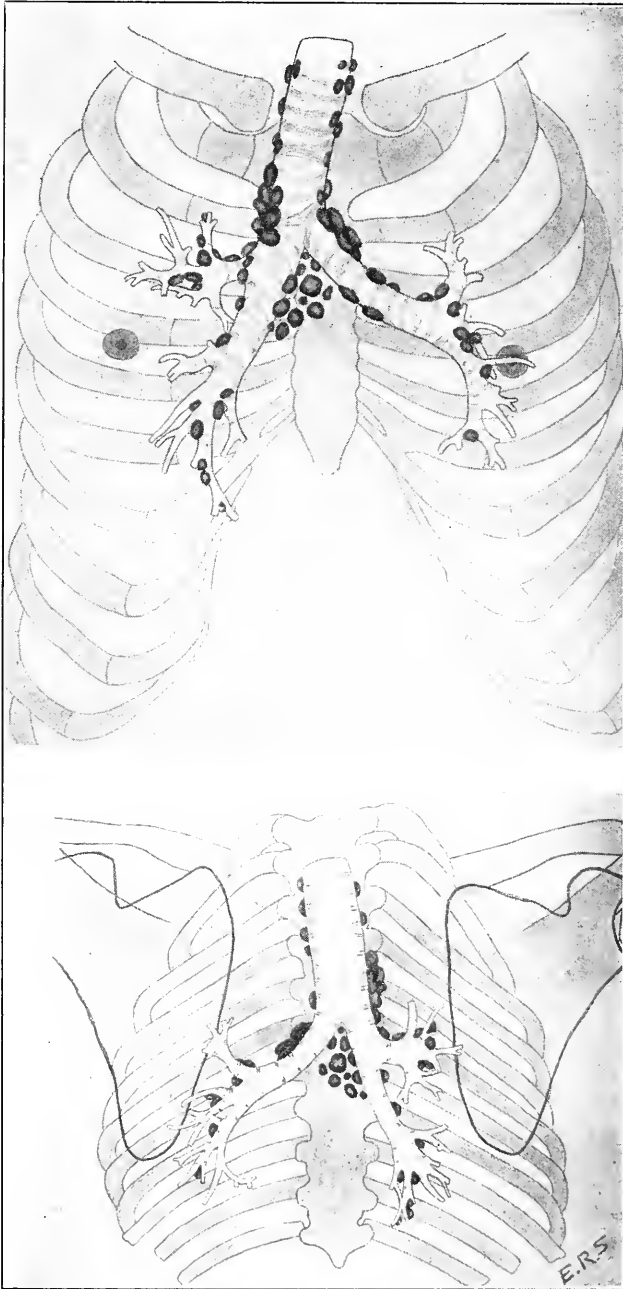
among 692 children of tuberculous parentage examined by the author, 469, or 67.8 per cent., had swollen cervical glands. A careful examination of children attending dispensaries shows that between 50 and 75 per cent. have palpable cervical glands. Most of them are due to carious teeth, hypertrophied tonsils, stomatitis, eczema or pediculi of the scalp, etc. That they are no indication that the intrathoracic glands are also swollen may be concluded when we bear in mind that anatomically the two groups have no direct connection, as has already been shown (p. 47).

Some distinction may, however, be made between enlarged cervical glands due to tuberculosis and those due to other causes. When the tumors in the neck are very large and persistent, showing little tendency to caseation and suppuration, they are almost invariably tuberculous. Of greater importance is enlargement of the supraclavicular glands, which drain the parietal pleura, especially when found unilaterally. This speaks for tuberculosis of the costal pleura, as has been pointed out by Hamburger. Ranke<sup>1</sup> has pointed out another characteristic of tuberculous cervical glands. They are apt to swell up at irregular intervals and retrogress again after remaining large for a few days or weeks, and each time the swelling increases there is an increase in the intensity of the constitutional symptoms. During the retrogression they become smaller, harder, lose their roundish contour and become fixed to the surrounding tissues. But while this sign is undoubtedly of value, it has failed me in several cases.

**Physical Signs of Tracheobronchial Adenopathy.**—The best that can be said about the physical diagnosis of tracheobronchial adenopathy is, that it is very indefinite; at any rate, all the criteria taken for proof of the existence of enlarged glands within the thoracic cavity do not enlighten us whether the process is active and demands active treatment, or is merely an innocuous enlargement of the glands of no clinical importance, as it actually is in the vast majority of cases. Judging by the anatomical relations of these glands, it is clear that they must attain some size before they become discoverable by percussion and auscultation of the chest. But that they often do attain large dimensions may be assumed when we consider the size attained by the cervical glands at times.

This group of glands includes those located around the trachea and bronchi, mainly in front of the bifurcation of the trachea. Pathologically, it has been found that those around the right bronchus are liable to attain very large dimensions and produce symptoms and signs of the disease. From the practical standpoint, in addition to the anterior and posterior mediastinal glands, there are three groups of glands which may become swollen because of tuberculous infection: At the bifurcation of the trachea we have the *tracheobronchial lymph nodes*; along the main bronchi there are the *bronchial lymph nodes*; and at

<sup>1</sup> Münch. med. Wehnschr., 1914, lxi, 2099.



FIGS. 73 and 74.—Composite drawings showing the relationship of the bronchial glands to the thoracic wall in the adult. The glands are according to Sukiennikow, and the trachea and bronchi are after Blake (*Amer. Jour. Med. Sci.*, 1899, cxvii, 320). In the child the trachea bifurcates at about the level of the intervertebral disk between the fourth and fifth thoracic vertebrae, which corresponds nearly to the tip of the fourth thoracic spine. This is about opposite the articulation of the third costal cartilage anteriorly. (Stoll.)

the hilus of the lungs there are the *pulmonary lymph nodes*, which also surround the bronchi, and communicate through lymph spaces with the parenchyma. In fact, all these glands receive their lymph from the pulmonary tissue and the bronchi. Considering their anatomical relations it is clear that when enlarged, they may exert pressure upon the bronchi and trachea, as well as on the nerves and bloodvessels passing through the chest. They may produce symptoms because of pressure exerted on the vagus and recurrent laryngeal nerves and the superior vena cava. They may even press upon the phrenic nerve, the arch of the aorta, innominate veins, etc. But this is exceptional despite the fact that text-books give so many signs revealing pressure on the various structures. The anatomical relations of these glands are shown vividly in the accompanying illustration (Figs. 73 and 74) from Stoll,<sup>1</sup> based on Sukiennikow's<sup>2</sup> anatomical researches.

*Inspection.*—On inspection the thorax is often found deformed in those who have had enlarged glands; indeed, some of the deformities produced by the intrathoracic glands are difficult to differentiate from the changes produced by early rickets. In some cases we find the typical phthisical chest, the *habitus phthisicus*—a long, narrow chest with the ribs slanting downward at an acute angle, and the intercostal spaces narrow. Children with such chests have passed through several attacks of glandular enlargement and may, at the time of examination, be in fair health. In many we see what Stoll calls the "hilus dimple." If the breath is held at the end of inspiration there is seen an apparent retraction on one or both sides in the second interspace. Owing to lack of expansion of one apex, the chest wall lags with inspiration. In old cases this "dimple" may remain permanently owing to permanent pleural adhesions or cicatrization of the peribronchial tissues at the hilus (Figs. 75 and 76).

This phthisical chest, which some authors consider predisposing to phthisis, is in fact proof that the patient has been tuberculous for a long time and in children it is proof that the thoracic glands have been enlarged. In our investigations of the form of the chest in children of tuberculous parentage, we found that at birth the chest is almost invariably normal, and only when tubercle affects the intrathoracic viscera are changes in its form produced. In some cases unilateral bulging of the chest wall is noted, especially the first two interspaces near the sternum.

Enlarged veins are often visible on a chest containing enlarged glands. They are usually seen on the upper part of the thorax, mostly bilateral though not symmetrical, and at times unilateral. In my own cases, 37.5 per cent. of children with tracheobronchial adenopathy had enlarged and visible veins on the thorax, and of these, three-fourths were unilateral. Of those in whom the diagnosis of latent tuberculosis

<sup>1</sup> Amer. Jour. Med. Sci., 1911, cxli, 83; *Ibid.*, 1914, cxlviii, 369; Amer. Jour. Dis. Children, 1912, iv, 333.

<sup>2</sup> Berl. klin. Wchnschr., 1905, xi, 316, 347, 369.

was justified, or in whom it was strongly suspected, 25 per cent. showed this sign, while among the manifestly healthy only about 1 per cent. had enlarged veins on the thorax. Stoll found enlarged and visible veins on the thorax in 92 out of 173 cases; of these 50 per cent. were tuberculous.

It thus appears that this is a fair sign of compression of the main trunks of the intrathoracic veins by enlarged glands or adherent pleura. My general experience, however, urges me against hasty diagnosis based on this sign alone. It is met with in many healthy children, especially such as have a delicate and transparent skin and also in anemia. In adults, it is often seen in women during lactation, when it may be unilateral, and in persons suffering from non-tuber-



FIG. 75

FIG. 76

FIGS. 75 and 76.—The "hilus dimple." (Stoll.)

culous affections of the bronchi, lungs and pleura, especially chronic bronchitis, asthma, and pulmonary emphysema (see p. 244).

*Percussion.*—A great deal has been written about percussion as an aid to the diagnosis of tracheobronchial adenopathy. But as a matter of fact there are many children with undoubted enlargement of these glands in whom the percussion note elicited over every part of the chest is practically normal. When we consider the topographical position of the bifurcation of the trachea, it is clear that the glands must become very large to produce dullness anteriorly or posteriorly over the surface of the chest. The various special methods like Koranyi's<sup>1</sup> vertebral

<sup>1</sup> Ztschr. f. klin. Med., 1906, lx, 295.

percussion, which has been elaborated in this country by John C. Da Costa,<sup>1</sup> do not give satisfaction. In many cases, however, there is found paravertebral dulness on light percussion. The areas that may be found affected correspond to the hilus—the interscapular space, especially the right, and anteriorly in the upper two interspaces near the sternum. To elicit this, very light percussion is necessary because of the thinness and resilience of the thoracic walls in the child. It may be found when the glands are not very much enlarged; then it is due to engorgement of the bloodvessels and lymphatics which exists in the region of the hilus during the acute stage. It is the collateral inflammation described by Tendeloo.<sup>2</sup>

This defective resonance is only rarely bilateral. Anteriorly it must be differentiated from the dulness due to an enlarged thymus. The latter is usually beneath the sternum, while in bronchial adenopathy the dulness is mainly at the side of that bone, mostly to the right. We must mention that there is normally an oval area of dulness between the first and fifth thoracic vertebræ, extending an inch to two outward on each side of the spine to which William Ewart<sup>3</sup> has called attention. But in cases of glandular enlargement it is usually unilateral—one interscapular space is dull. I have seen a few cases in which enlarged thoracic glands produced dulness all over one side of the chest. Another point is that this dulness, to be indicative of adenopathy, must be permanent, found during several examinations. As has been pointed out by Grancher and J. E. H. Sawyer,<sup>4</sup> in debilitated and rachitic children there are observed transient areas of dulness due to a bronchus being plugged with secretions and the resulting atelectasis of the air vesicles it supplies.

*Auscultation.*—In my experience auscultation has been of more service in attempting to diagnosticate intrathoracic glands. In children the breath sounds are louder and somewhat harsher than in the adult—puerile. But this, in healthy children, is heard all over the chest. Enlarged glands alter them in circumscribed areas. Thus when large we may find feeble breathing over a limited area owing to compression of a bronchus, or to modifications in the pulmonary circulation in that region. On rare occasions the breath sounds are feeble over an entire lung anteriorly and posteriorly. But this is liable to great fluctuations. I have followed some children for years and found that at times there are modifications in the breath sounds in a given area which shift so that at the next examination one or more months later, the modification is found at another place. It may be found that during an attack of an intercurrent disease, rhinopharyngitis, influenza, etc.—when the glands swell up and there is an exacerbation of the tuberculous process—the auscultatory phenomena make their appearance to disappear after the acute process is gone.

<sup>1</sup> Amer. Jour. Med. Sci., 1909, cxxxviii, 815; 1913, cxlvi, 660.

<sup>2</sup> Sixth Intern. Congr. on Tuberculosis, 1908, vi, 197.

<sup>3</sup> Brit. Med. Jour., 1912, ii, 966.

<sup>4</sup> Birmingham Med. Review, 1912, xix, 57.

Anteriorly the auscultatory signs in children are uncertain, because normally we may hear the tracheal sound at the sides of the manubrium in emaciated children with narrow chests. Still, when tubular breathing is heard unilaterally at the side of the sternum it speaks for enlarged glands. Posteriorly, bronchial or harsh breathing in the interscapular space of one side is an indication of the transmission of the tracheal murmur by enlarged glands which act as sound conductors. In mild cases only prolonged expiration is heard in one interscapular space, but in those in which the glands are very much enlarged, the breathing over a limited area may be tubular or exquisitely bronchial, almost the same as is audible when listening directly over the trachea.

**D'Espine's Sign: Tracheophony.**—About twenty-five years ago A. d'Espine<sup>1</sup> described a sign of enlarged tracheal glands which is more satisfactory than any other symptom or sign. It consists in auscultation of the voice, especially the whispered voice, along the course of the trachea posteriorly. He described this sign as follows: The patient is told to count "one, two, three," or "thirty-three," as clearly as possible (younger children may be told to say "papa," "mamma") while the examiner auscultates with the naked ear, or better with a stethoscope, the spines of the cervical vertebræ. As long as we listen to the cervical spines, we hear the characteristic tracheal tone and each word is quite clear. In the normal child this clear voice stops abruptly as soon as we reach the seventh cervical spine and the lung begins; but in cases with bronchial adenitis the clearness of the voice or the tracheal tone continues lower down from the first to the fifth thoracic vertebra. It is at this spot that the main localization of the enlarged bronchial glands is found. The transmission of the tracheal tone in these cases is effected by the enlarged glands which surround the trachea at its bifurcation and often reach the spinal column, acting as sound conductors between the trachea and spine.

When auscultation of the full voice gives uncertain results, the patient is told to whisper "thirty-three," which is even more reliable than the bronchophony just spoken of. It must always be borne in mind that in healthy children and adults, the bronchophony and the whispered voice stop abruptly at the seventh cervical spine, and when heard lower it is a sure sign of something interposing between the trachea and the spine, and acting as a voice conductor.

This sign of tracheobronchial adenopathy has been extensively tried in France and many report that it is more reliable than any other sign. Barot<sup>2</sup> found it superior to percussion and even more trustworthy than skiagraphy for the purpose of ascertaining the presence or absence of enlarged thoracic glands. In this country it

<sup>1</sup> *Traité des Malad. de l'enfance*, Paris, 1900, p. 856.

<sup>2</sup> *Arch. médicales d'Angers*, 1907, xii, 18.



has been strongly recommended by Stoll, Sewall,<sup>1</sup> Howell,<sup>2</sup> Honeij,<sup>3</sup> and others.

In evaluating this sign it must be borne in mind that the height of the bifurcation of the trachea, where the glands are most likely to become enlarged in tuberculosis, differs according to the age of the patient. In infants and young children it is on a level with the seventh cervical vertebra. But with advancing age it sinks lower and lower. At the age of eight it reaches the second or third thoracic vertebra and at twelve it is found as low as the fourth. In adults, especially in senile individuals, it may be found as low as the fifth or sixth thoracic vertebra. Therefore, in a child of ten the transmission of the whispered voice to the third thoracic vertebra may not mean enlarged glands in the chest.

It must also be emphasized that the mere transmission of the vocal resonance as heard over normal lungs is not d'Espine's sign. This is found very often in children without enlarged glands. It is the transmission of the characteristic tracheal timbre which counts. In most cases it is heard not only along the spine, but also in the interscapular space on one side; at times bilaterally.

I have tested this sign in various ways and found it most satisfactory. In several cases the skiagraphic plate failed to disclose the presence of enlarged glands while d'Espine's sign revealed them. Armand Dellile,<sup>4</sup> Zabel,<sup>5</sup> and d'Espine mention cases which were verified by autopsy.

**Smith's Sign.**—Eustace Smith's sign of bronchial adenopathy remains to be mentioned. It consists in this: If the child be made to bend back the head, so that the face becomes almost horizontal, and the eyes look straight upward at the ceiling above him, a venous hum, varying in intensity according to the size and position of the diseased glands, is heard with the stethoscope placed upon the upper bone of the sternum. As the chin is now slowly depressed, the hum becomes less loudly audible and ceases shortly before the head reaches its ordinary position. Smith explains this phenomenon in this fashion: While the head is bending backward, the lower end of the trachea is tilted forward, carrying with it the glands lying in its bifurcation, and the left innominate vein, as it passes behind the first bone of the sternum, is compressed between the enlarged glands and the bone.

In my own experience this sign is not very reliable. It is found in short-necked children without enlarged glands, and is absent in many with adenopathy. Gibson<sup>6</sup> pointed out that it is mostly found in children who have enlarged veins in the neck and on the chest.

<sup>1</sup> Jour. Amer. Med. Assn., 1913, lx, 2027.

<sup>2</sup> Amer. Jour. Dis. Child., 1915, x, 90.

<sup>3</sup> Jour. Amer. Med. Assn., 1913, lvii, 958.

<sup>4</sup> Diagnostic et traitement de l'adenopathie trachéo-bronchique, Paris, 1911.

<sup>5</sup> Münch. med. Wehnschr., 1912, lix, 2664.

<sup>6</sup> Brit. Med. Jour., 1906, ii, 1051.

**Reflex Symptoms.**—There are other symptoms of tracheobronchial adenopathy which are described in great detail in text-books, but which are in fact very rare and may be left out of consideration in the average case. Thus, pressure on the recurrent nerve may produce paralysis of the right vocal cord; pressure on the sympathetic may produce differences in the size of the pupils. Pressure on the vagus may produce tachycardia and palpitation, transient or permanent. But these symptoms are very rare and are not conclusive even when encountered.

In young children caseated glands may break through into adjoining structures, the bronchi, trachea, esophagus, etc. More rarely yet, the swollen glands acquire such dimensions that by pressure on a bronchus they prevent the entry of air into the part of the lung supplied by this tube; or by pressure on the trachea fatal asphyxia is produced. But these cases are extremely rare and may be considered medical curiosities.

**Skiagraphy.**—With the enthusiasm of the first years of radiography, we thought that with the aid of the  $x$ -rays we had at last found a means for positively identifying enlarged tracheobronchial glands. Radiographers often made diagnoses of tuberculosis in children who showed no symptoms of active disease and continued well indefinitely.

This was but natural, considering that normal glands allow the rays to pass through without casting any shadows, unless there is engorgement. Caseated glands cast a shadow which is occasionally distinct, but at times very indefinite. Only calcified glands cast a distinct shadow which may be identified in the vast majority of cases. But calcified glands, tuberculous in origin undoubtedly, are an indication that the disease has come to a standstill; in fact this is the only mode of cure of caseated glands.

Under the circumstances the most easily diagnosticated cases of tracheobronchial adenitis, when the  $x$ -rays are used for the purpose, are those which have no significance clinically—those with calcified glands. When we attempt to clear up a case in which the glands are swollen but neither caseated nor calcified—in other words, at a time when therapeutic measures may be inaugurated with a good chance of helping the patient—the  $x$ -rays very frequently fail to give conclusive proofs of the existence of trouble. On the other hand, they show old and calcified glands which may not be, and often are not, the cause of the clinical symptoms for which the patient consults us at the time.

Fluoroscopy is of no value at all in most cases of young children who cannot be managed in a totally dark room, and asked to breathe deeply, cough, etc. The best is a skiagraphic plate taken instantaneously and studied after it is developed. But even here we must be careful before concluding that because there is a shadow at the hilus, there is active tuberculosis of the intrathoracic glands. In nearly all infectious diseases of childhood, but especially in scarlet fever, measles and whooping cough, these glands are enlarged, but the

swelling slowly retrogresses during convalescence. In fact de Mussey attributed the paroxysms of cough in pertussis to enlarged glands. It is therefore wrong to diagnose tuberculous adenitis in a child with whooping-cough, or scarlet fever, as I have seen done.

Sluka<sup>1</sup> insists that several plates taken at long intervals are necessary, so that evanescent enlargements of the glands may be excluded. In fact he found that the shadows shown on the plate of the same child at irregular intervals have been larger at one time and smaller at another; at times involving almost a complete lobe, or even a whole lung, at other times only a small circumscribed shadow was found; at one time in the right side, at other times in the left, etc. A considerable part of these changes is due to changes in the collateral inflammation in active cases, but it seems to me that differences in the technic of taking the picture, the distance of the tube from the patient's chest, the sharpness of the focus, the condition of the tube, etc., are responsible in many cases.

On the whole there is no doubt that shadows in the region of the hilus are indicative of enlargement or engorgement of the glands in that region. This mottling and stippling of the hilus is, however, no criterion as to the activity of the disease. Even the triangular or wedge-shaped shadow, with the base to the hilus, which has been described by Stoll and Heublein, Sluka and others, is no proof of active disease, as the writer has repeatedly convinced himself. It appears also that in young infants these hilus shadows are only rarely seen even when adenopathy exists. Sluka says that in children under two years of age he never obtained a shadow on a chest plate which would even remotely suggest hilus tuberculosis, though he has taken numerous plates of sick children. He says that only during the third and fourth year do the glands begin to reveal themselves roentgenologically; they are mostly seen during the sixth and seventh years, and then begin to decrease in frequency.

Of late the confidence formerly placed in *x*-ray findings in intrathoracic conditions has been waning. At the 1915 meeting of the American Pediatric Society,<sup>2</sup> Koplik said that "one should be very cautious in permitting an *x*-ray to make a diagnosis for him." Holt stated that he had "sent the same case to a radiologist on successive days and each day a different diagnosis was made. The *x*-ray is very misleading and a dubious procedure upon which to base a diagnosis."

In doubtful cases the skiagraphic plate may give some indefinite information about the presence of enlarged thoracic glands. But when found in a child showing no clinical symptoms of the disease, we must not conclude that the child is actively tuberculous. We do not as yet have enough experience with skiagraphy in healthy children, nor have enough autopsies been made to verify skiagraphic findings, to warrant unequivocal conclusions.

<sup>1</sup> Wien. klin. Wchnschr., 1913, xxvi, 254.

<sup>2</sup> Medical Record, 1915, lxxxviii, 502.

**Tuberculin Diagnosis.**—Basing their opinion on the fact that tuberculosis in infants is almost invariably fatal, it has been concluded that when in a young child any of the tuberculin tests is positive, and there are some symptoms, such as cough, etc., the child should be pronounced tuberculous to the great dismay of the parents. I have seen children kept from school and thus deprived of an education, and perhaps hampered for the rest of their lives, solely because the von Pirquet reaction was found positive.

We have already shown that the tuberculin reaction shows but one thing—whether the person—child or adult immaterial—has ever been infected with tubercle bacilli. But it does not show conclusively whether the infection was followed by disease. Inactive infection is more likely to give a strong reaction than active tuberculous disease. In fact, in fatal tuberculous bronchopneumonia, meningitis, etc., the reaction is negative; in others it is but faintly positive. In other words, the stronger the reaction, the less likelihood of active or dangerous disease in the child, and a negative reaction is no positive proof of the absence of dangerous tuberculous disease.

In infants under two years of age a positive reaction is to be taken as an indication of active disease because at that age infection is very likely to be followed by disease; during the first six months of life, almost invariably. But after two years of age harmless infections are the rule, so that the value of the tuberculin reaction acquires an academic importance, as was already shown, but it loses its clinical value. This is a point which pediatricists should bear in mind. It should never be lost sight of that after the third year latent tuberculosis is very common and this gives the same reaction as active disease.

**Diagnosis.**—The diagnosis of tuberculous tracheobronchial adenopathy depends on the presence or absence of clinical symptoms of disease. A child over two years of age showing a three plus tuberculin reaction, and a shadow in the region of the hilus on the skiagraphic plate is to be considered well and healthy as long as it presents no symptoms of disease; as long as there is no fever, no cough, no emaciation, etc. It is different with those who have clinical symptoms. In these it is always important to remember that when a child does not thrive, fails to gain in weight, the cause must be found. If it is not found, and there is cough, especially that dry, brassy cough, the temperature is to be taken three or four times a day. If it is found that there is an irregular fever, of the type described above, there is presumption of tuberculosis. If on examining the chest we find some dulness in one of the interscapular spaces or anteriorly in the upper two interspaces near the sternum; and the whispered voice and the tracheal tone along the spine, and in one or both interscapular spaces are audible in the peculiar characteristic fashion described when speaking of d'Espine's sign, the diagnosis of tracheobronchial adenopathy is clinched.

It is different when these signs are found, even in conjunction with

skiagraphic findings and a positive tuberculin reaction, in a child which shows no clinical symptoms of disease. There is no doubt that this child may also have, and probably does have, enlarged bronchial glands. But these glands are not actively diseased, and as long as the little patient thrives, there is no cause for alarm. The glands are of no more clinical value than the scars found in the apices of 90 per cent. of adults who die from causes other than tuberculosis; they are of no more serious import than the enlarged glands found on the necks of over 50 per cent. of evidently healthy children in the slums of large cities.

**Prognosis.**—The prognosis of tuberculosis in children under ten years of age embraces two problems: (1) The immediate outlook; and (2) the ultimate outlook. In other words, what are the chances of survival or of retaining good health immediately after infection has taken place, and is the child destined to develop phthisis after reaching the age of adolescence?

The immediate outlook appears to be good, provided the lesions remain localized in the glands or even in the bones and joints. This is clearly seen in cases of superficial glandular tuberculosis: Most children with tuberculous cervical adenitis, especially those requiring no operative interference, recover after a protracted illness. The same is true of osseous and articular tuberculosis. From 900 cases of tuberculous disease of the hip treated by A. Bowlby<sup>1</sup> at the Alexandra Hospital in London during twenty-one years, 33 died—a mortality of 4 per cent. He found that of the 33 who died, 24 were attacked by the disease before the age of six. The mortality from tuberculous tracheo-bronchial adenitis is undoubtedly even lower. The greatest danger is metastasis in the meninges, but even this is comparatively infrequent after the fifth year.

For this reason all methods of treatment of tuberculosis in children produce most excellent results. This is also the reason why orphan asylums—which harbor children between four and fourteen years of age—report that, despite the fact that most of their inmates are derived from the poorest strata of population and an enormous proportion are of tuberculous stock, they have no morbidity nor mortality from tuberculosis. It is simply because death from tuberculous tracheobronchial adenopathy is extremely rare. The success of the open air schools, the preventorium, etc., should also be attributed in a great measure to this cause.

Barring meningeal complications, or intercurrent diseases, the prognosis in tracheobronchial adenopathy is excellent.

In older children, seven years of age or more, the prognosis of apical pulmonary tuberculosis of the same type as seen in adults, is more serious, though not as serious as in adults. It appears that pulmonary lesions in children heal with greater ease than in adults, though now and then we meet with a case in which the process in the

<sup>1</sup> Brit. Med. Jour., 1908, i, 1465.

lung proceeds to cavitation and the child succumbs to the usual clinical manifestation of phthisis. After the twelfth year there is hardly any difference in the clinical picture and prognosis of phthisis in children and in adults.

Says Franz Hamburger,<sup>1</sup> one of the most experienced men in this field: "In general we can lay down the fundamental principle that the prognosis of tuberculous manifestations in children is not at all bad. It is, in fact, one of the most important achievements of recent years that we know: 'tuberculosis in children is a relatively harmless disease.' It will naturally take decades till the lay public will learn this important fact." And I may add till physicians in general will learn it.

The prognosis also depends on several other factors: The younger the child showing active tuberculous manifestations, the worse the outlook, the more liable it is to suffer from, or to succumb to, metastatic tuberculous manifestations, such as meningitis, rupture of a gland into a bronchus, the trachea or esophagus. These complications in fact become less frequent after the third year of life, and after the sixth year they are comparatively rare. The prognosis also depends on various accidental complications. Thus, a child that escapes the endemic diseases, such as measles, whooping cough, scarlet fever, diphtheria, etc., may grow up into healthy manhood in spite of the enlarged glands in the chest which disappear in nearly all cases after the tenth year; at any rate they no more give trouble. It is thus clear that the prognosis also depends on the social and economic conditions under which the child is raised. Those who are well off in this regard survive unscathed, because they have good nourishment, healthy dwellings, frequent vacations and are less likely to contract other diseases, etc.

The second element in the prognosis of tuberculosis during childhood is the problem whether every child infected at an early age is destined to become phthisical after the fifteenth year of life? The facts observed in daily practice seem to be against such a view. If this were the case tuberculosis among adults would not kill only one out of seven to ten individuals, as is now found wherever there are available vital statistics, but over 90 per cent. of humanity would succumb to phthisis. That an active tuberculous lesion during childhood is not necessarily followed by phthisis in later life is evident from the following facts:

We meet with many persons showing unmistakable signs of having had some form of tuberculosis during childhood, but pass through life as healthy and even vigorous individuals. This is the case with those showing scars on the neck which are undoubtedly remnants of tuberculous adenitis which had suppurated or were operated upon. We meet with many showing remnants of articular and osseous tuber-

<sup>1</sup> In Brauer, Schröder, and Blumenfeld, *Handbuch der Tuberkulose*, 1915, v, 31.

culous disease, yet they pass through life without developing phthisis. In fact the contrary seems to be true. Those who see large numbers of phthisical patients are struck by the fact that consumptives with scars on the neck, or with ankylosis of joints following earlier tuberculosis, etc., are extremely rare. This point, which has already been touched upon (see p. 112), seems to indicate that an early tuberculous lesion may have some immunizing effect on the organism and prevent the development of phthisis in later life.

We are, at the present state of our knowledge, not warranted in asserting that this protection against phthisis conferred by a early tuberculous disease depends on infection with bovine tubercle bacilli, as some have been inclined to assume. But we may safely draw a conclusion that an early tuberculous disease of the tracheobronchial glands is not necessarily followed by phthisis in later life, and there seems to be evidence that it may act in the same manner as articular, osseous, and glandular tuberculosis.

## CHAPTER XXV.

### PHTHISIS IN THE AGED.

**Frequency.**—While discussing the frequency of tuberculosis during the various age periods we have shown that no age is exempt; in fact it appears from available mortality statistics that after the age of twenty the death-rates from phthisis are about the same till very advanced life. While making autopsies pathologists are often struck with the frequency with which active tuberculous lesions are found in the lungs of aged persons, and investigations in homes for the aged show clearly that a large proportion suffer from phthisis.

The reason why popular opinion has ascribed immunity of old subjects to phthisis appears to lie in the fact that, when occurring, this disease runs a mild, benign course and may pass off as bronchitis, asthma, etc. But when the sputum expectorated by senile persons is examined, it is very frequently found to contain large numbers of tubercle bacilli. In fact these aged consumptives may be considered actual bacillus "carriers" who, without themselves suffering very much from the bacilli, disseminate the disease much more widely than younger patients who know of their condition and the dangers of indiscriminate expectoration.

**Etiology.**—Most phthisis in the aged has been acquired during childhood, but has been held in abeyance throughout life, to break out again at the period of life when the organs of the body begin to suffer as a result of wear and tear. Others have suffered from some form of phthisis before, but the disease was "cured," to reawaken during old age. Many have been afflicted for years with some form of fibroid phthisis, but when senile degeneration began to manifest itself the tuberculofibroid lesions in the lungs began to activate with more vigor.

From our present knowledge of phthisiogenesis we must exclude new infections of aged persons, because they have been infected during the earlier years of life, as was already discussed elsewhere. A new or primary infection in an adult would surely not pursue such a slow, sluggish course as is seen in the aged. The active disease in senile individuals should be considered either metastatic or else old, perhaps dormant processes flaring up and causing disease.

Pathologically, there are no differences in the lesions between the aged and those in adults in general, with but few exceptions. In the aged the fibroid processes predominate because the tendency to fibrosis of tissues is characteristic of advancing age. These fibroid formations tend to limit the lesions, prevent its spread and to surround the cavities,



which show no tendency to enlarge by contiguity of the process. On the other hand, bronchiectatic cavities are more frequently found in old than in young consumptives.

**Symptoms.**—"The conditions with which it may be associated modify the course of the tuberculous process," says J. Edward Squires,<sup>1</sup> "so that the symptoms are obscured and the signs of its presence in the lung are somewhat indistinct. Tuberculosis, when it attacks lungs already damaged by the degeneration of age, may add but little to the discomforts of the individual who is already short of breath and 'wheezy.' The increasing infirmity of the patient is accepted as a sign that he is aging more rapidly, and no suspicion of any added mischief is aroused or entertained." Generally speaking the symptoms of phthisis in the aged are often those of fibroid phthisis, which have already been described. From most patients who consult us for hemoptysis, cough, expectoration, and a lesion is discovered on physical examination, we elicit a history that they have been troubled with some of these symptoms for years, perhaps since childhood, but that they have been considered as suffering from chronic bronchitis or pulmonary emphysema.

The patients cough, but the cough is mild. In aged persons the stimulus for cough is not as intense as in the young because the sensibility of the bronchial mucous membrane is greatly diminished. The quantity of sputum they expectorate is, as a rule, not very considerable because they have a tendency to swallow it. When told that they are tuberculous they are apt to resent the imputation claiming that they have coughed for years, perhaps since they can recall, and if it had been "consumption" they would have succumbed long ago.

Most senile patients are of slim build, but occasionally we meet with a tuberculous patient over sixty who is above the average weight. But with the onset of active symptoms they begin to lose in weight, and within a few months they may be reduced to mere skeletons.

A large proportion of patients have no fever, though the methodical use of the thermometer per rectum may reveal a typical tuberculous temperature with slight rises, to 101° F. in the afternoon. In this respect phthisis does not differ from other diseases in the aged. We know that pneumonia may pass an afebrile course in the senile. The organism of the aged does not react with fever as does the body of the young.

The pulse is more rapid than normal for the age of the patient. In rare cases tachycardia is seen, especially when there is cardiac displacement. Dyspnea is a frequent symptom, especially after exertion. Because of the concomitant arteriosclerosis and myocarditis, cyanosis is not uncommon. In the later stages, when heart failure is apt to occur, edema of the extremities is frequently seen. The blood-pressure is low considering the age and the condition of the arteries of the

<sup>1</sup> International Clinics, Sixteenth Series, 1906, iv, 90.

patient. Hemoptysis occurs quite frequently. In most cases it is merely streaky sputum, but it may be profuse and I have seen a fatal hemorrhage in a woman, aged seventy-eight years. Nightsweats are rare because with advancing age the sweat glands undergo atrophy, and also because the great oscillations of temperature characteristic of phthisis in the young are absent in the senile.

**Physical Signs.**—The appearance of the senile phthisical chest depends on the character of the lesions in the lungs. In those in whom there is pulmonary emphysema in addition to the tuberculous process there is the characteristic barrel-shaped chest, rigid, hardly expanding, in fact always in the position of maximum inspiration. All that is seen is that the entire chest is lifted up with each inspiration, but there is no anteroposterior or lateral expansion. The intercostal spaces are wide and the direction of the ribs is more horizontal than normal. But many have no old emphysema and in them the thorax is rigid owing to the ossification of the costal cartilages; the ribs run at a more acute angle to the spine than normal and the intercostal spaces are wider; the supraclavicular and infraclavicular spaces are deeply excavated, more so on one side. During fits of coughing either apex or both, may be seen blowing up in the supraclavicular space. Dilatation of the veins of the neck is a frequent symptom, and when there is relative tricuspid insufficiency, owing to dilatation of the right heart, there may be a venous pulse. Kyphosis and kyphoscoliosis is never absent.

Auscultation is also not as satisfactory as in young subjects. The breathing is superficial and, combined with pulmonary emphysema which is only rarely absent, we may hardly hear any breath sounds, or only a feeble murmur is audible. These are also the reasons why bronchial or cavernous breathing are so rarely heard over the sites of cavities. Bronchovesicular breathing of low pitch, with prolonged expiration may, however, be made out over one apex at times while carefully auscultating the chest. Rales are not audible in many cases because of the superficial breathing; but over the sites of excavations large consonating rales may be heard even when no breath sounds are made out. At the base these rales are usually due to bronchitis or bronchiectasis which are very frequent in old age.

**Course.**—In many cases the cough, expectoration, emaciation, etc., continue for years and, inasmuch as these old persons do not follow occupations necessitating physical exertion, the true nature of the disease is not even suspected. They are considered patients suffering from chronic bronchitis or emphysema. I know several old consumptives who have survived children and grandchildren whom they infected with tuberculosis. In fact, whenever I discover children with signs of tuberculous infection, though a history of exposure cannot be made out, I inquire for the grandparents, and have on many occasions found that one of them was the source of infection, though he did not know the true nature of his illness.

In the vast majority of cases the tendency of the disease is to progress, though slowly, and never to a cure. Occasionally we find that it advances rapidly, assuming an acute or subacute course, with hectic fever, rapid emaciation, etc. Owing to the weakness and the general debility the cough is usually not at all severe, and when there is no fever, a diagnosis of carcinomatosis is made. Others cough and expectorate for years, when suddenly fever develops and the patient is carried off within a few days. Bronchopneumonia may have been erroneously considered the cause of death, unless the sputum was examined and tubercle bacilli were found; a diagnosis of acute primary tuberculosis may then be erroneously made. Darenberg speaks of acute phthisis in the aged, and Hoppe-Seyler speaks of acute miliary tuberculosis on rare occasions. But these cases are evidently acute exacerbations of chronic phthisis which had been kept in abeyance for years.

**Diagnosis.**—The diagnosis is not difficult when the possibility of phthisis is kept in mind in all cases of cough, expectoration, emaciation, etc., met with in senile patients. Most of the mistakes made in these cases are due to failure to examine the sputum for tubercle bacilli. When the physical signs in the chest are indefinite, which is often the case, the bacteriological findings decide. When looking for fever in these cases we should never rely on the axillary temperature; only the rectal is to be taken.

We must guard against mistaking signs of old, healed lesions for active disease. This can be avoided by a careful study of the symptomatology and bacteriology of the affection.

## CHAPTER XXVI.

### COMPLICATIONS OF PHTHISIS.

Most of the conditions described as complications of phthisis are part and parcel of the tuberculous disease in the chest, or symptoms of the disease which at times assume the ascendancy. This is the case with hemoptysis, ulcerations and amyloid degeneration of the intestines, bronchitis, tuberculosis of the kidneys, etc. These have been discussed more or less while speaking of the symptoms of phthisis. There are, however, left a few of the more important complications which deserve separate description. Among these pleurisy, spontaneous pneumothorax, laryngeal, intestinal, and meningeal tuberculosis, etc., are the most important.

**Dry Pleurisy.**—Inflammation of the pleura cannot be considered a complication of phthisis in the strict sense of the word, because it is part and parcel of the tuberculous process in the lung, and often manifests itself earlier than the symptoms of the disease, as was already shown.

During the course of phthisis the patient often complains of pains in the chest which last for several days, a week or two, and then pass away, to return sooner or later in the same or another area. Auscultation may reveal friction sounds, etc. These frictions are mostly heard over the lower parts of the thorax, especially in the anterior axillary line. But pleural inflammation may occur during the course of phthisis without causing pains in the chest.

This form of dry pleurisy is not always to be considered an ominous complication of phthisis. In fact it is a rather salutary phenomenon in many cases. The fibrosis it causes in the pleura overlying the tuberculous lesion in the lung, as well as the adhesions of the pleural layers which are formed as a result of it, at times obliterating the pleural cavity, prevent the tuberculous lesion in the lung from breaking through into the pleura. Without adhesive pleurisy spontaneous pneumothorax would be a very frequent complication of phthisis. It appears to be an expression of the efforts of nature to limit the progress of the disease; it limits the excursions of the affected part of the lung, thus affording it rest which is essential for a cure. It is also an indication that the organism responds to the tuberculous infection with a productive inflammation, with the formation of connective tissue, which is the main element in the cure of the disease.

**Pleurisy with Effusion.**—Less frequently encountered during the course of phthisis is moist pleurisy. But it may occur at any stage of the disease. When occurring at the onset of the disease, its course

is that of the usual attack of pleural effusion, excepting that the patient must be watched carefully for the development of a lung lesion after the effusion has been absorbed or aspirated, as has already been mentioned.

In the advanced stages of the disease, pleural effusions, serous or purulent, may occur, at times filling the greater part of the affected side of the chest. Localized, encapsulated and interlobar effusions are also found at times. In fact, because the pleura is more or less obliterated in most advanced cases, effusion when occurring can only be localized. These small effusions may cause no special symptoms, and if the patient is not examined frequently and with minute care, they are overlooked. Skiagraphy has recently shown the large number of small effusions that otherwise escape notice.

In many cases a copious effusion, even if serous, is a serious complication, but in some its effects are either negative, or altogether beneficial. The lung is compressed in the same manner as with the gas introduced when inducing a therapeutic pneumothorax, and is thus given an opportunity to heal. The writer has met with several cases of phthisis in which a serous effusion has thus favorably influenced the tuberculous process in the lungs.

An exception is to be considered a hemorrhagic effusion into the pleura which in itself is to be considered characteristic of either tuberculosis or cancer. In the former the fluid is only slightly blood-stained, while in the latter the proportion of blood may be considerable. In phthisis sanguineous effusions are of serious prognostic importance. Purulent effusions are always ominous.

The onset of pleurisy with effusion during the course of phthisis is in most cases acute, with pain and tenderness in the inframammary and axillary regions, increased by cough and deep breathing. The temperature, in cases which were afebrile, rises to 101° F. or more, and keeps up high for several days, coming down slowly to its former level. In many cases, however, the onset is insidious, with vague pains in the chest, or altogether painless. We often find signs of dry pleurisy or of an effusion in phthisical patients without any history of an acute onset.

The physical signs of localized dry pleurisy are friction sounds and a decrease in the intensity of the breath sounds over the affected area. The friction sounds are shuffling in character, at times creaking or squeaking. In some cases they greatly simulate crepitation or even rhonchi. The criteria for their differentiation given in text-books—persistence after cough, superficial localization, and increase on pressure with the stethoscope, etc.—are often inadequate to distinguish them. Perhaps the fact that they are heard during both phases of respiration is the best distinguishing sign, but even this is at times misleading.

When effusions occur the breath sounds are altogether absent or very feeble, and flatness is found on percussion. Small effusions are

more easily discovered in the left side when finding that Traube's semilunar space is dull. But in chronic phthisis this is not to be taken without qualification because thickened pleura in that area may give dullness. On the other hand, small effusions are often found in the left side leaving Traube's space resonant. Grocco's triangle of paravertebral dullness is made out in most cases. Localized empyemata are best made out by the aid of skiagraphy.

Serous effusions may be absorbed within a few weeks, but in many cases they remain for months or indefinitely.

The effects of pleural effusions on the course of phthisis have been extensively discussed of late. In former times they were considered very unfavorable. Lately several authors have pointed out that an effusion into the pleura may be rather of good augury, collapsing the lung and bringing it to rest, as we attempt to do when inducing artificial pneumothorax. It appears, however, that the whole problem is not so much about the effusion, excepting when it is very copious and menacing, which is rather rare, but depends on the underlying pulmonary lesion. When the latter is unilateral, not extensive, and shows no tendency to progressive activity, an effusion usually has no influence at all. These effusions appear and disappear without injuring the patient. But when the lesion is active, extensive and progressive, a pleural effusion may do considerable harm; especially is this true when the effusion occurs in the side of the chest harboring the minor lesion, while in the opposite side there is extensive cavitation, etc.

**Empyema.**—Empyema may occur during any stage of phthisis, though it is a comparatively rare complication. It seems to me that in most cases in which it occurs there was a spontaneous pneumothorax which was not recognized. Of late it is frequently encountered in the form of pyopneumothorax after the induction of therapeutic pneumothorax. It may be localized, interlobar, and then it is very difficult of diagnosis, the symptoms passing as part and parcel of the phthisical process. The microorganisms found in the pus include tubercle bacilli, pneumococci, and the pyogenic microorganisms. The kind of microorganism found has no influence on the symptomatology, course, and outcome of this complication.

The onset of empyema during the course of phthisis is usually insidious. In fact many cases are altogether latent and discovered accidentally, or with the  $x$ -rays. The latency is best explained by the fact that the usual symptoms of empyema are such as are met with during the course of phthisis without this complication. Moreover, occurring as it does in the more or less advanced stages of the disease, the purulent effusion takes place into a pleura which is adherent, and therefore the process remains limited. In fact it is very frequently sacculated. Because the lung has been damaged by the tuberculous process, it has lost a great part of its expansive power, and will not easily reexpand after removal of the pus, and the prognosis is thus very unfavorable.

Empyema is one of the most dangerous complications of phthisis. Spontaneous absorption never occurs. Operations for the removal of the pus are very unsatisfactory. The result is usually that the fever, cachexia, and amyloid degeneration of the viscera carry off the patient sooner or later. I have had 2 cases in which empyemata have opened into the bronchi and the pus was expectorated. The patients were "cured" of the empyema, but the tuberculous process proceeded its course.

**Gangrene of the Lungs.**—This is an exceedingly rare complication of phthisis; it is more often found in cases of bronchiectasis, especially in old subjects. Considering that mixed infection is very frequent in phthisis, although the contaminating microorganisms are not responsible for most of the symptoms of the disease, it is surprising that putrefactive germs should but rarely take root in phthisical lungs. When occurring it is soon recognized by the fetid breath and expectoration. But not all phthisical patients with fetid sputum have gangrene of the lung. Sputum retained in tuberculous cavities may become fetid. But in such cases the malodorous expectoration lasts only for a few days or weeks and sooner or later assumes the odor usually met in phthisis. Its odor also is different from that of gangrenous sputum—it is of a sweetish and nauseating character, while in gangrene it is pungent and actually suffocating. The constitutional symptoms in gangrene are characteristic: The temperature is raised high, the patient passes into a septic state with acute asthenia, and succumbs rapidly. In afebrile cases of phthisis a sudden rise in the temperature accompanied by fetid sputum is a sure indication of complicating gangrene of the lung.

**Spontaneous Pneumothorax.**—This is the most frightful complication of phthisis. It is of more significance than copious hemorrhage because the latter only terrifies the patient and its ultimate prognosis is usually favorable, as we have already shown, but pneumothorax is deadly and the victim is justified in his apprehension that the collapse and agonizing dyspnea are indications that he is breathing his last. From West's<sup>1</sup> statistics it appears that 70 per cent. of patients attacked by pneumothorax die, and in phthisis the proportion is even higher.

"To anyone carefully examining the lungs of patients dying of acute phthisis," says Williams,<sup>2</sup> "the marvel is not that pneumothorax should occur, but that it should not do so more frequently, for it is not uncommon to see what appears to be several abscesses immediately underlying the visceral pleura, and apparently ready to burst." Chronic lesions are usually well protected against bursting into the pleura by more or less massive adhesions, and pneumothorax mostly occurs in acute cases in which the process extends quickly before adhesions can form.

<sup>1</sup> *Dis. of the Organs of Respiration*, London, 1909, p. 837.

<sup>2</sup> *Pulmonary Consumption*, London, 1887, p. 206.

**Frequency.**—The frequency of this complication varies with the character of the clinical material. It is not very frequent in hospitals for advanced cases because only old cases are admitted in whom pleural adhesions prevent its occurrence. According to Powell, about 6 per cent. of the fatal cases of phthisis at the Brompton Hospital, in London, succumbed with pneumothorax; Williams found 10 per cent. and Weil even 13 per cent. In many cases sudden death in phthisis is caused by this complication, though it may not have been recognized. At the Montefiore Home we meet with about a dozen cases annually among 300 treated. As has been pointed out by Sir Douglas Powell, the lesion is more likely to occur in the left than on the right side. From a collection of 234 cases reported by Louis, Walshe, West, and himself, he finds that in 95 it was on the right and in 139 on the left side. Powell attributes it to the greater frequency with which the left lung becomes the seat of tuberculous disease.

**Symptoms.**—The onset is sudden, unexpected. The patient has known that he is tuberculous for some time, and may have been assured that his prospects for an ultimate recovery are good. But suddenly, like a thunderbolt out of a clear sky, after a fit of coughing, some slight exertion, or without any exciting cause at all, he is seized with a sharp agonizing pain in the chest, he feels as if "something has given way," or as if something cold is trickling down his side. He at once sits up in bed holding his hand fast over the affected side, gasping for breath. Acute distressing dyspnea, cyanosis, a small, rapid and feeble pulse, cold, clammy extremities and other phenomena of collapse soon make their appearance. The facial expression is that of profound agony, the eyes prominent, the lips livid and the forehead clammy. The respirations are frequent—fifty or more per minute, and superficial. The temperature, which may have been elevated for some time, suddenly drops to below normal and the cough, which may have been annoying before the accident occurred, ceases for a time; perhaps because of the pain the patient restrains himself.

In very acute cases the patient may expire within a few hours as a result of profound shock, dyspnea, and heart failure. Many of the cases of sudden death in phthisis are due to this cause. But in most cases the circulation adapts itself by degrees to the altered conditions of the thoracic viscera, the dyspnea is ameliorated, the temperature rises to above normal and the patient feels somewhat relieved, the air hunger not being as acute as at the onset, though he still breathes forty or more times per minute, and is still cyanosed. Within a few days, usually between the third and fifteenth day an effusion of fluid into the affected pleura is found, hydropneumothorax, or pyopneumothorax.

The size of the perforation into the lung has but little influence on the acuity of the distress—a small opening the size of a pinhead may permit the entry of sufficient air into the pleura to collapse the lung completely and to displace the thoracic and abdominal organs just as



## PLATE XIV

FIG. 1



Dense infiltration of the upper half of the left lung with displacement of the heart to the left. Right lung emphysematous.

FIG. 2



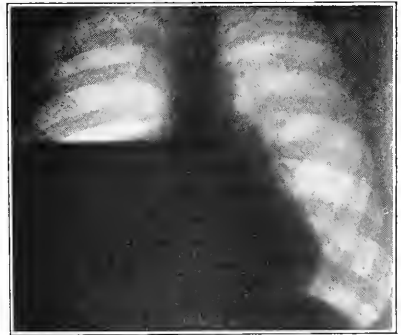
From same patient as Fig. 1. Spontaneous pneumothorax, air filling left pleural cavity, and displacing the heart to the right.

FIG. 3



Pneumothorax in right pleura extending in a thin layer of air from the diaphragm to the apex. Right lung slightly collapsed and presents consolidation at its lower third. The rest appears studded with cavities and calcified nodules. Lower half of left lung emphysematous; upper half nodular infiltration, especially at axilla. Heart and trachea displaced to the right.

FIG. 4



Hydropneumothorax in the right pleura.



well as a larger one. In fact, in some quickly fatal cases only a small opening or slit is found at autopsy, while in others with large openings little distress is seen, healing is rapid, and the patient may last for months. At the necropsy it is found that the opening is usually small, linear, slit-like, and occasionally circular, at times attaining the size of a dime. In some cases there are two or even three perforations.

**Varieties of Pneumothorax.**—It is of clinical significance whether the perforation closes speedily and no more air or pus can pass into the pleural cavity, thus allowing absorption of the air. The symptoms, prognosis, and treatment depend mainly on this point. There are described in text-books three varieties of pneumothorax—*open*, *closed*, and *valvular*. In the open variety there is a patent opening which permits air to pass in and out of the pleural cavity and the tension within the affected pleura is equal to that of the external air. In the closed variety the perforation has healed, and the air in the pleural cavity may be absorbed sooner or later, as is the case with induced therapeutic pneumothorax, with or without the development of an effusion which is generally serous. In the valvular variety, during inspiration or cough air enters freely into the pleura, but is prevented from coming out again during expiration by a valve or contraction of the slit. The result is that the tension within the pleural cavity becomes very high and, pushing the mediastinum to the opposite unaffected side, causes distressing dyspnea, cyanosis and heart failure till the patient is no longer able to cope with the situation and succumbs.

This interpretation of pneumothorax has of late been questioned by West, Bard, Castaigne and others. West says: "All pneumothorax is at first valvular, at any rate more or less, *i. e.*, the air finds more or less difficulty on expiration. Thus the pleura becomes more and more full of air and the lungs more and more compressed, and this obviously tends to close the hole more or less completely. When the hole is of ordinary size, it will become patent on inspiration and thus admit air, but only so long as the pressure in the pleura is less than that of the air in the air tubes. As soon as the pressure on the two sides is equal, no more air can enter, and the hole remains closed. If the edges cohere, the hole will remain permanently closed; if not, as soon as the pressure in the pleura is diminished, as it may be by paracentesis, the orifice may open again into the pleura. This is the explanation in many cases of the return of dyspnea after paracentesis."

As has been pointed out by Sir R. Douglass Powell, the displacement of the mediastinum is not necessarily due to the pressure exerted by the air in the pleural cavity. His manometric measurements have revealed no positive pressure in pneumothorax. From his investigations he is inclined to believe that the dislocation of the heart is due to the unopposed traction exerted by the elastic unaffected lung. Because they are no longer held up by the elasticity of the lung, the diaphragm and the abdominal viscera sink downward.

*Partial Pneumothorax.*—In old chronic cases of phthisis we meet with partial pneumothorax in which there is a perforation into the pleural cavity, but owing to dense adhesions the air is only filling up a limited pouch at a place where the pleural sheets are not adherent. The onset is less acute and the symptoms of collapse are usually absent. The patient may have some pain in the chest, dyspnea, etc., but these attract little attention in a disease like phthisis in which these symptoms are so frequent without the occurrence of pneumothorax. Careful physical examination may disclose signs of the condition, but it is easier to find it out with the aid of skiagraphy. I have seen cases in which skiagraphy could not decide. It is often mistaken for a large cavity, especially when it is localized over an apex, but even in the lower parts of the chest it may exquisitely simulate pulmonary excavation.

*Latent Pneumothorax.*—At times we meet in tuberculous patients a pneumothorax without a history of an acute onset with pain, dyspnea, collapse, etc. In some of these cases careful inquiry elicits a history pointing to a subacute onset, but such symptoms are quite common in chronic phthisis without this complication. In one case admitted to the hospital we found complete collapse of the lung and we at first suspected an artificial pneumothorax, produced before admission, but it turned out to be a latent case.

In chronic phthisis we also meet with cases in which there is a sudden onset with all the symptoms of this accident, but physical examination fails to reveal any of the signs. The French call it *pneumothorax muet*, the silent form. In these cases the signs do appear, however, within a few days. In one of my cases of this character a radiographic plate showed that the air was filling the thoracic cavity for an inch or two along the axillary line. In others there was an interlobar air pouch. These forms are best diagnosed with the *x*-rays.

*Double Pneumothorax.*—Double pneumothorax has been met with in phthisis on exceedingly rare occasions. It is incompatible with life. But D. Hellin<sup>1</sup> and R. Staehelin<sup>2</sup> mention cases which lasted for days.

**Physical Signs.**—The affected side of the chest is larger—in the maximum inspiratory position; the shoulder raised, the intercostal spaces obliterated, tense and tender to the touch. While the number of respirations is fifty or more per minute, movements of the thorax are seen only in the unaffected side, while the affected side is fixed, almost immobile. In the vast majority of cases the apex beat cannot be seen, but when visible it is found at the left axillary line in right-sided pneumothorax and at the xyphoid cartilage or even beyond it in left-sided perforations. Vocal fremitus is abolished over the affected side.

Instead of the dull note which was found before the accident, the

<sup>1</sup> Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1907, xvii, 414.

<sup>2</sup> Mohr and Staehelin's Handbuch der inneren Medizin, Berlin, 1914, ii, 756.

## PLATE XV

FIG. 1



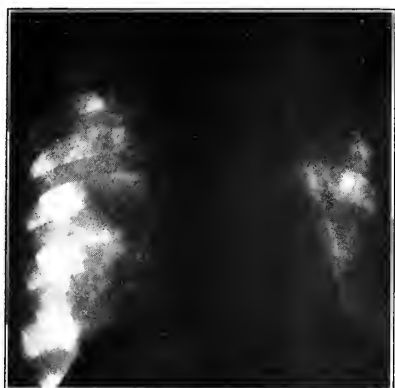
Left pleura filled with air, but large cavity with dense walls under second and third interspace did not collapse. Nodular infiltrations throughout right lung. Dilated bronchi and enlarged glands in hilus region.

FIG. 2



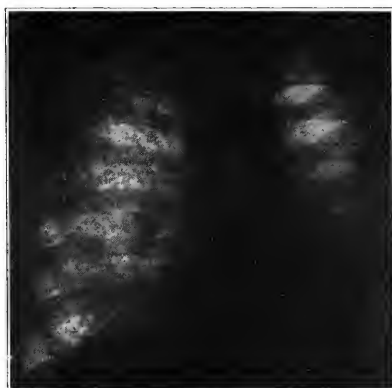
Complete pneumothorax of right thoracic cavity pushing mediastinum to the left and compressing the left lung. Trachea visible as markedly displaced to left.

FIG. 3



Old fibroid phthisis with extensive involvement of the left lung and pleura. Spontaneous pneumothorax of right pleura.

FIG. 4



Diffuse tuberculous process all over both lungs; marked peribronchial infiltrations, and calcified glands along the hilus. The apex is infiltrated and adherent in the left side; below the clavicle there is a circumscribed pneumothorax, which on physical exploration gave signs of a cavity. The lower half of the left pleura is thickened, which cannot be differentiated in the radiogram from fluid.



affected side emits a hyperresonant, sometimes a tympanic note, depending on the tension of the air within the pleural cavity. By comparison, the unaffected side appears to emit a defective or dull note. In cases in which the upper part of the pleura is adherent and does not collapse, the apex is dull or "boxy" on percussion. When later fluid makes its appearance in the pleural cavity, we elicit a flat note over the lower part of the chest and the flatness changes its level with the change in the patient's position. Shifting dulness is pathognomonic of air and fluid in the pleural cavity. Displacement of the thoracic and abdominal viscera can be made out more or less easily by percussion. In right-sided lesions the liver dulness disappears altogether, or is displaced downward, and the heart is shifted to the left; even as far as the axillary line; in left-sided pneumothorax the heart dulness may be completely absent, or displaced to the right, and the splenic dulness may also be absent. In fact the spleen and the liver may be felt distinctly low in the abdomen. The displacement of the heart may be noted a few minutes after the occurrence of the accident.

We may also elicit various metallic or amphoric notes on percussion, especially with a coin placed over the chest and tapping it with a stick or pencil, while listening with the naked ear or stethoscope over the opposite side of the chest. Biermer's and Wintrich's signs, as well as cracked-pot resonance may be elicited in many cases.

Auscultation shows complete absence of breath sounds over the affected side of the chest in cases in which the opening is small or closed and the lung is completely collapsed. When the upper parts of the pleura are adherent, the auscultatory phenomena of the original lung lesion are audible, but below no sounds at all are heard. But in most cases there are heard amphoric breath sounds at some point between the shoulder-blades. Exceptionally we meet with a case of pneumothorax in which the voice and breath sounds are audible in an exaggerated form all over the affected side. When the opening into the lung is large, permitting the passage of air from the bronchi into the pleural cavity, we may hear an exquisite variety of amphoric breathing or metallic sounds which are characteristic. The voice sounds, as well as the cough, may also have a metallic echo.

The splashing or succussion sound is audible at a distance in many cases, and the patients themselves are annoyed by it. Some patients know how to jerk their bodies to produce it to the best advantage. I have had patients in whom the succussion sound was the only indication of fluid in the thorax, all other signs being absent because of the depression of the diaphragm, the result of the pressure exerted by the tension of the air in the pleura. It is an excellent proof of the existence of air and fluid in the pleura. It is stated that it may be elicited in the stomach and colon, but I have not met with a case in which this vitiated a diagnosis.

**Metallic Tinkling.**—A clear musical note, heard at intervals on listening over a hydropneumothorax, resembling a drop of water falling into

a reverberating vessel, may be heard in some cases. At times it is only heard after cough. It is apparently not due to the falling of a drop at all, but to a rale produced in some portion of the lung which acquires a metallic character by reverberation.

**Diagnosis.**—The diagnosis of pneumothorax has undergone quite some changes within recent years since we have had an opportunity to study this condition produced artificially in tuberculous patients for therapeutic purposes, and also since we employ skiagraphy for the purpose of examining the chest. We now have explanations for some phenomena which were formerly obscure, and we know that certain signs formerly considered pathognomonic of pneumothorax, are not at all invariable accompaniments of the disease.

In the usual case of pneumothorax during the course of phthisis, the sudden onset of urgent dyspnea, pain in the chest, collapse, etc., coupled with physical signs of pulmonary collapse, suffice to establish the diagnosis. But there are many sources of error. We may have pneumothorax without any of these acute symptoms, as has been already stated. In fact, since the *x*-rays have been employed the number of latent and silent cases of pneumothorax has enormously increased. On the other hand, we meet in phthisis cases of acute dyspnea, pain, and even collapse not due to this accident. Especially difficult are the cases of partial pneumothorax, because the mediastinum is not displaced, and a thickened pleura may obscure the tympany and the absent or amphoric breath sounds may be otherwise interpreted. At times it is very difficult to differentiate a partial pneumothorax from a large pulmonary cavity, and before the advent of skiagraphy mistakes of this kind were more frequently made than at present. The differentiation is usually of practical value, because the prognosis in cases with large excavations is very unfavorable, while with a localized pneumothorax it is more hopeful.

Even in cases with complete collapse of the lung, tympany may not be elicited on percussion, as we have learned lately in cases of artificial pneumothorax. It appears that it all depends on the tension of the air within the pleural cavity. In hydropneumothorax, tympany is found when there is but little fluid and considerable air; but when the effusion is copious we get flatness which disappears when the fluid is aspirated, provided the pleura is not too thick.

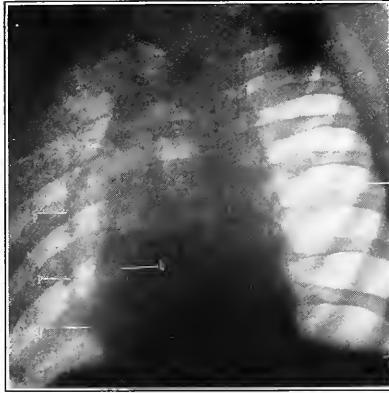
The position of the heart is usually of assistance in deciding whether we deal with a large cavity or a pneumothorax: In the former it is displaced toward the affected side, while in the latter it is moved away from it. But even here there are many important exceptions owing to previous pleural adhesions, etc. Skiagraphy usually decides, but not always.

The signs obtained on auscultation differ very much in cases of open as compared with closed pneumothorax, and in the latter cases it depends on whether the lung is completely or only partially collapsed. A closed pneumothorax with complete collapse is silent; no breath



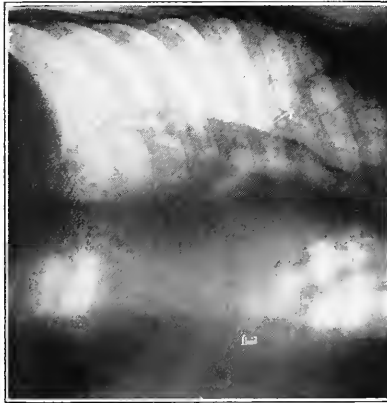
# PLATE XVI

FIG. 1



Spontaneous pneumothorax complicated by hydro- or pyopneumopericardium. Shows pneumopericardium and beginning effusion into pericardium; pneumothorax in left pleura. *p*, pericardium; *pn*, pneumothorax; *fl*, fluid in chest; *h*, heart.

FIG. 2



Hydropneumothorax; pneumopericardium; patient lying on the left side. The fluid in the left pleura shifted to the axillary side. Air in the right side of the pericardium is plainly visible. (Case of Dr. A. Meyer.)



sounds at all are audible as a rule. At times we perceive some bronchial breathing in the interscapular space emitted from the bronchi near the spine. In the open variety we usually hear amphoric breathing of an exquisite type. In many cases of phthisis, in which the pleura is free all over, it is adherent at its upper third, over the site of the main lesion, and does not collapse at that place, and we obtain the breath sounds and rales peculiar to the diseased lung.

The breath sounds often audible over a completely collapsed lung were formerly attributed to some opening into a bronchus allowing air to pass in and out of the pleura. We now know that this is not always the case because in artificial pneumothorax, where an opening into the lung is positively excluded, we often perceive the same acoustic phenomena. It seems that the air in the pleural cavity is capable of transmitting the sounds in the bronchi when in a certain condition of tension.

The bell sound is almost invariably heard in all cases in which the effusion is not too thick, as in some cases of pyopneumothorax. It is easily elicited by placing a coin over the anterior surface of the thorax and percussing it with another while auscultating posteriorly or in the axilla. A clear, ringing, bell-like sound, which is characteristic, is heard. But exceptionally it is also heard over large cavities, or even a dilated stomach. It is often absent in pneumothorax; but when heard it is of significance, showing, as it does, air and fluid in the pleural cavity. We may hear it only with the patient in the horizontal position. In some it appears only after some of the fluid has been aspirated.

A positive diagnosis of pneumothorax can be made when one is alert and looks for it in every suspicious case. In most cases the abrupt onset of the urgent symptoms and the physical signs suffice. In doubtful cases the Roentgen rays decide easily and speedily.

A rare complication of pneumothorax, the spontaneous as well as the artificial varieties, is pneumopericardium—air entering the pericardial sac. We then have instead of the cardiac dulness, hyperresonance or tympany, sometimes cracked-pot sound. On auscultation we hear that the heart sounds are extraordinarily intensified, and a splashing sound is audible, or a succussion sound, synchronous with the systole. In the case observed by the author there was also a metallic tinkle and a friction fremitus, especially when the patient bent his body forward. Similar cases have been reported by Wenckebach,<sup>1</sup> Cowan,<sup>2</sup> Harrington and Riddell, and Alfred Meyer.<sup>3</sup> With the aid of skiagraphy the diagnosis offers no difficulty, as can be seen from the skiagram (Plate XVI) of Dr. Meyer's case.

**Prognosis.**—On the whole, the prognosis of spontaneous pneumothorax is decidedly gloomy. Occurring, as it does, in patients who are

<sup>1</sup> *Ztschr. f. klin. Med.*, 1910, lxxi, 402.

<sup>2</sup> *Quarterly Jour. of Med.*, 1914, vii, 165.

<sup>3</sup> *Medical Record*, 1915, lxxxviii, 991.

usually doomed because of the condition of the lungs, this accident but accelerates the inevitable. In very acute cases the patients succumb within a few days, and 90 per cent. die within a month. An open pneumothorax, permitting the entry of the contents of pulmonary cavities into the pleura is almost invariably fatal sooner or later.

While there have been reported cases of hydro- and pyopneumothorax that have survived for years and some in which the fluid has been absorbed, they are exceedingly rare and in all cases that I have seen the patients succumbed within one year after the onset of the complication.

Conditions are somewhat different with the closed cases of pneumothorax, also the partial variety. They usually occur in patients with but slight tuberculous lesions and with good resisting power. As long as there is no communication with a tuberculous cavity, and the pleura is not infected, as is the case with artificial pneumothorax, the air in the pleura may in time be absorbed. In fact, it was these rare cases of collapse of the lung and the resulting amelioration of the symptoms of phthisis, which suggested the idea of therapeutic pneumothorax, of which we will speak later on.

**Laryngeal Tuberculosis.**—The frequency of this complication during the course of phthisis has been differently stated by various authors. The proportion varies from 5 to 50 per cent. Harold Barwell found at the Mount Vernon Sanatorium 11.69 per cent. among 1541 tuberculous patients; Brandenburg, 9.16 per cent.; John B. Hawes,<sup>1</sup> only 8 per cent. among 1245 patients. Even sanatoriums, which do not admit patients with laryngeal complications, have many with this disorder. Thus at Otisville, N. Y., Julius Dworetzky<sup>2</sup> found that 25.6 per cent. had laryngeal tuberculosis. It seems that the proportion found depends on the zeal displayed by the laryngologists looking for it. Percy Kidd<sup>3</sup> found that 50 per cent. of fatal cases of phthisis showed tuberculous laryngitis at the autopsy, and of these only 20 to 50 per cent. were recognized during life. The estimate that one out of three patients with active phthisis has a laryngeal lesion, appears to be correct.

Laryngeal tuberculosis spells phthisis; primary tuberculosis of this organ is so exceedingly rare as to constitute a medical curiosity. It is more frequent among males than among females, the proportion being, according to Morel Mackenzie, 2.7 of the former to 1 of the latter. The reason for this disparity is that men are altogether more liable to throat affections, probably because of the abuse of tobacco, alcohol, and exposure to irritation by dust at their occupations. It is also likely to be more severe in men than in women.

**Symptoms.**—These depend on the location of the lesion in the larynx. Those in whom the interior of the larynx is affected do not suffer as

<sup>1</sup> Boston Med. and Surg. Jour., 1914, clxxi, 19.

<sup>2</sup> Ann. Otology, Rhinology and Laryngology, 1914, xxiii, 835.

<sup>3</sup> Allbutt's System of Medicine, v, 210.

much as those whose trouble lies at the entrance of the larynx. The symptoms are few in number. Hoarseness is present in all in whom the interior of the larynx is affected, and it may be of various degrees, from mild tiring of the voice to complete aphonia. On the other hand, pain is more frequent when the entrance of the larynx, especially the epiglottis is affected, while the voice may in these cases be retained quite well. The pain may be spontaneous, radiating to the ear, or there may be a sensation of tickling which provokes cough. In advanced cases, with perichondritis, deep ulceration of the epiglottis and collateral inflammatory edema of the parts, the pain may be so severe as to interfere with swallowing food. Usually warm fluids and solids cannot be passed. The dysphagia may be so severe as to prevent swallowing altogether. I have seen some cases in which swallowing



FIG. 77.—Tuberculosis of the larynx. (Ballenger.)

of saliva was more painful than that of food. Local external tenderness is rare. Stridor and obstruction of respiration are comparatively rare, but they do occur now and then.

**Diagnosis.**—Considering the immense prognostic significance of laryngeal tuberculosis, we must be guarded in making a diagnosis of this complication. Hoarseness alone is insufficient for a diagnosis because it may be absent when the larynx is implicated but the vocal cords remain in good shape; or it may be present in a patient suffering from phthisis, yet no tuberculous lesion is discoverable in the larynx. This is seen when the right recurrent laryngeal nerve is implicated in a thickened right apical pleural lesion, or when the two laryngeal nerves are pressed upon by enlarged tracheal glands. It must also be borne in mind that simple chronic laryngitis and pharyngitis are extremely common in phthisical subjects, as has been pointed out by Harold S. Barwell,<sup>1</sup> and they may cause hoarseness and throat discom-

<sup>1</sup> *Lancet*, 1909, i, 1249.

fort. The constant coughing and the irritation of the sputum passing through the larynx may produce a simple laryngeal catarrh.

W. Freudenthal<sup>1</sup> urges that lasting hoarseness apparently due to simple laryngitis, and seen in a patient who is not presenting symptoms of alcoholism, or constitutional diseases as gout or rheumatism, should excite suspicion of tuberculosis.

The diagnosis of tuberculous laryngitis is quite easy when there are ulcerations but in the incipient stage it appears to be just as difficult as the diagnosis of incipient pulmonary tuberculosis. Laryngologists usually enumerate the laryngoscopic signs of advanced disease, evidently because they mostly see advanced cases.

Some authors have maintained that the tuberculous larynx is characterized by pallor of the mucous membrane. But it appears that pallor alone is insufficient for a diagnosis because the larynx shares the pallor of the fauces which is seen in most tuberculous patients; it is also found in those who suffer from severe anemia of any kind. In fact, there are just as many red and congested larynges in phthisical subjects as pale ones.

Paresis of the vocal cord on the side of the lung lesion, associated with slight chronic laryngitis, is one of the signs of incipient tuberculosis of the larynx, according to many authors, notably F. Stern.<sup>2</sup> He calls this the "larynx sign" of early pulmonary tuberculosis and advises direct visual inspection to detect it when there is a sensation of vague oppression of the chest, a tendency to rheumatic pains, slightly irregular breathing or gastric disturbances. The entrance to the throat is moderately red and the paralyzed vocal cord is also red. There is always more mucus on the paretic cord than on the other, and its inner margin is usually irregular in outline. There is slight hoarseness, particularly at night and the patient hawks often but raises very little sputum and tubercle bacilli may not be found at this early stage.

Minor,<sup>3</sup> whose opinion is of value because he could follow his cases both from the general clinical, and the laryngological findings, states that when a laryngeal catarrh begins to localize itself and becomes unilateral, it is suspicious. Next to this, he considers highly significant a grayish wrinkling of the posterior commissure and a table-like elevation of its mucous membrane. Early changes are also found in the processus vocalis, the posterior insertion of one cord, or the body of the cord itself.

Ulcers of the cord, instead of being localized in one spot may be scattered along the edge, producing the characteristic nibbled-out appearance, but in most cases the posterior end of the cord is red, beefy and thickened. Thickening and yellowing of the false cords or ventricular bands are other early changes mentioned by Minor,

<sup>1</sup> *Ztschr. f. Tuberkulose*, 1910, xvi, 338.

<sup>2</sup> *Berl. klin. Wochenschr.*, 1914, li, 1419.

<sup>3</sup> *National Assn. Study and Prev. Tuberc.*, 1910, vi, 186.

and also localized congestions or anteroposterior thickenings of the arytenoids.

Thickening and even ulceration of the posterior wall of the larynx is another early sign. Uniform redness of both vocal cords is not pathognomonic of tuberculosis, but when one cord is red while the other remains normal or is pale, tuberculosis is probably present.

With the advance of the process the smooth and shiny appearance of the parts is changed owing to the ulceration. The infiltration often affects the epiglottis, producing that pale, rounded, sausage-like body

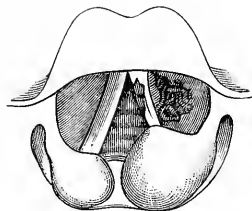


FIG. 78.—A tubercular ulcer on the left ventricular band and left vocal cord. Pear-shaped edematous swelling of aryepiglottic folds, more intense on the side of the ulceration. (Cohen.)

which may attain such dimensions as to obstruct the view of the interior of the larynx. The arytenoid cartilages often change into pyriform bodies. When the infiltration begins to ulcerate, the characteristic worm-eaten appearance of the parts is seen, together with caries, perichondritis, necrosis, and exfoliation of parts of the cartilages.

In cases in which the infiltration begins in one or both vocal cords or the ventricular bands, or the interarytenoid region, the prognosis



FIG. 79.—Tubercular infiltration of the interarytenoid space with tubercular papillomata of both vocal cords. Characteristic edematous infiltration of the aryepiglottic folds. (Cohen.)

is more favorable. However, one or both cords may be destroyed by ulceration. In far advanced cases all parts may be destroyed, including the epiglottis, of which only a short stump may be left.

**Prognosis.**—The outlook in phthisis complicated by tuberculous laryngitis is rather gloomy, though not invariably fatal, as was once thought. Thirty-five years ago Morell Mackenzie stated that “it is not certain that any cases ever recover.” His statistics showed that it reduced the average expectation of life to twelve or eighteen months, very few patients living more than two and a half years. But since

phthisis has decreased in malignancy during recent years, patients suffering from laryngeal tuberculosis have also benefited and we now know that many recover. The lesion in the throat may heal, as has been found by careful studies of postmortem findings.

The laryngeal lesion *per se* only rarely kills the patient and it has been stated that consumptives never die from the larynx. This is wrong, of course, because we occasionally see a case of sudden death from asphyxia, or edema of the glottis. The bulk of the patients with laryngeal complication die as a result of the severity of the pulmonary lesion, or inanition due to painful deglutition. In fact, when the larynx is extensively involved, producing dysphagia, dysphonia, etc., a fatal issue may be expected sooner or later. If the lesions in the lung and larynx are not sufficient to kill the patient he will die as a result of inanition.

The milder forms of laryngeal tuberculosis have a better outlook. Many heal spontaneously without any local treatment. The general treatment instituted often hastens recovery from the laryngeal lesion. Very often the condition of the larynx goes hand-in-hand with the general condition of the patient, both improving, or aggravating simultaneously. Others are benefited by local treatment.

**Tuberculous Ulceration of the Intestines.**—The frequency of intestinal ulcerations found at autopsies on tuberculous subjects would indicate that they are more frequent than they are diagnosed *intra vitam*. Thus Louis found ulcers in five-sixths of his cases; Bayle and Lebert in two-thirds; Williams found at the Brompton Hospital postmortems in 81 per cent. intestinal ulcerations of a tuberculous nature; and Percy Kidd found them in 71 per cent. While they are responsible for the diarrhea in advanced phthisis in most cases, in many it is due to the toxemia, the toxic substances in the blood being eliminated through the intestines, or swallowed sputum is the cause. Lardaceous disease of the intestines is very frequently responsible, while errors in diet, especially an excess of fat or of milk, may induce diarrhea which is difficult to control.

There may be eight, ten, or even twenty motions a day, expelling loose, dark or chocolate-colored matter, exceedingly fetid, and it may contain small sloughs from the bowels. Quite often it is tinged with blood, but copious hemorrhages from the bowel are exceedingly rare. John M. Cruice<sup>1</sup> says that when they do occur it is of grave prognostic significance. The first case of this kind was reported by Tonnelle in 1829. In 1892 Guyenet could find only 15 cases in medical literature and Cruice found 10 additional cases in 1913. Although the prognosis is very grave in intestinal hemorrhage, Peters, Bullock and Bonney report cases that recovered.

One characteristic of tuberculous diarrhea is its persistence. It may be checked by proper dietetic and medicinal treatment, but no

<sup>1</sup> Medical Record, 1913, lxxxiv, 471.



sooner is this omitted than it reappears. With the diarrhea the emaciation proceeds at a rapid pace and they usually foreshadow quick relief from the suffering. I have seen patients who have been gaining, lose within one week all they have gained in months, and within two to four weeks they were reduced to mere skeletons.

**Diagnosis.**—It is very difficult to say with certainty whether a diarrhea in a consumptive is due to toxemia or to intestinal ulceration. Tenderness is often found in the right illiac fossa, but it may be all over the abdomen or any part of it. J. Walsh<sup>1</sup> made a thorough study of the symptomatology of intestinal ulceration, comparing it with autopsy findings in 100 cases at the Phipps Institute. The usual symptoms relied on—diarrhea, and abdominal pains, tenderness and rigidity, especially in the region of the ileocecal valve—were carefully studied. He found that singly these symptoms add little or nothing to the diagnosis of intestinal tuberculosis, nor do any two, or all four when found in the same patient, because they may be encountered while the autopsy shows no ulcerations in the intestines and the reverse. The presence of an ischiorectal abscess in an advanced case adds to the probabilities of intestinal ulcerations. Nor has he found any relation between the presence or absence of albumin in the urine, or the results of the diazo-reaction, or indican in the urine, and intestinal ulceration. He concludes that the diagnosis of intestinal tuberculosis cannot be made with the slightest degree of certainty from our present known symptoms, and since the condition carries with it such an unfavorable prognosis, he advises that it is best that the diagnosis should not be made, so that the patient will have a better chance for hopeful treatment.

While the outlook for healing of these ulcers is remote, yet it is possible. Amenomiya<sup>2</sup> shows that regeneration and healing are possible, even without scar formation, but the muscular coat is never regenerated.

**Peritonitis.**—The pathogenicity of tuberculous peritonitis as a complication of phthisis is no more the disputed problem which it was formerly. Considering the frequency of bacillemia in phthisis, it is clear that the blood may bring tubercle bacilli to the peritoneum just as readily as to other serous membranes. It is not as frequent a complication as is laryngeal or intestinal tuberculosis, but it apparently occurs more often than is suspected at the bedside, and we are at times surprised to find it at the autopsy when *intra vitam*, even in carefully watched cases, it was not suspected.

Authors disagree as to its frequency in phthisis. Münstermann<sup>3</sup> found it in 5 per cent. of cases; Börschke<sup>4</sup> in 16.17 per cent. In his autopsy material P. Horton-Smith Hartley found it in only 3.4 per

<sup>1</sup> National Assn. Study and Prev. Tuberc., 1909, v, 217.

<sup>2</sup> Virchows Archiv, 1910, cci, 231.

<sup>3</sup> Die Bauchfelltuberkulose, Munich, 1890.

<sup>4</sup> Virchows Archiv, 1892, cxxvii, 121.

cent. of cases. Perforation of tuberculous ulcers of the bowels were observed in 3 cases out of 263 autopsies, or a percentage of 1.1, the perforation in each of the instances occurring in the ileum. It appears to be very frequent in acute miliary tuberculosis, but in chronic pulmonary tuberculosis it is less often encountered. While in many cases the infection of the peritoneum can only be explained by assuming that the bacilli were brought there by the blood, in a considerable number they may travel by way of the lymphatics from the pleura, the pericardium, from the mesenteric lymph glands and above all by contiguity from infiltrated Peyer's patches and ulcers of the intestines. They may also come by contiguity from tuberculous lesions of the urogenital system, especially from the adrenals which are often the seat of tuberculous changes in phthisis.

**Symptoms.**—We meet mainly with two forms of this complication: dry, adhesive, and moist or exudative, both of which may be acute or chronic. During the course of phthisis the acute form, in the clinical sense, is usually due to perforation of an intestinal ulcer, or more rarely a pyothorax breaking into the peritoneal cavity, when it may produce suppurative peritonitis. In one case in which during life the condition was not even suspected, I found at the autopsy a minute opening through the diaphragm permitting leakage of the pus from a pyo-pneumothorax. Fenwick<sup>1</sup> maintains that in some cases there may be premonitory symptoms, viz., pain for a few days before actual perforation takes place from a tuberculous intestinal ulcer; in others there may be bilious vomiting, the abdomen is distended, and hyperresonant on percussion. These premonitory symptoms are obviously due to local acute peritonitis. The actual perforation may occur during straining at stool, during an attack of vomiting or retching, or altogether while the patient is at rest. Some patients feel an acute pain or a sensation as if something had given way in the abdomen. Collapse ensues and within a few hours or days the patient succumbs to cardiac failure. Some recuperate from the shock but they succumb within a few days to the symptoms of acute peritonitis, or more rarely to exhaustion.

The chronic form may be overlooked because it often runs its course symptomless. The patient may complain of abdominal pain, vomit and have diarrhea, but these symptoms are very frequent during the course of phthisis without any peritoneal complication. On the other hand, there are cases with peritonitis in which all these symptoms are lacking. The ascitic form is exceedingly rare in phthisis, though now and then we meet with a case in which the abdomen is filled with fluid. To be sure there are many cases with exudates, but they usually escape detection until they assume large dimensions—1500 c.c. of fluid in the peritoneal cavity may be in the pelvis, etc., and not be discovered by ordinary examination.

<sup>1</sup> *The Dyspepsia of Phthisis*, London, 1894, p. 176.

The adhesive form is characterized by the formation of adhesions and cicatricial contractions of the mesentery and gluing together loops of the gut are very frequent. Especially frequent are adhesions of the peritoneum to the liver and spleen. The adhesions and cicatricial contractions at times produce incomplete stenosis of the intestine with resultant persistent constipation and uncontrollable vomiting. Colicky pains increased by pressure and on movement may be observed. In these cases the emaciation may be extreme despite the fact that the local lesion in the lungs is not extensive, nor very active. When the inflammation in the peritoneum is limited and circumscribed, which is not infrequent, the pain may be localized at one point. It is noteworthy that fever may be absent, but in most cases of active phthisis, pyrexia due to the lung lesion is so frequent that it cannot be utilized for diagnostic purposes as to the presence or absence of a peritoneal complication. On the other hand, when the lesion in the lung is quiescent or latent, the complicating peritonitis may pass an apyretic course. In many cases there is diarrhea due to intestinal catarrh or, more frequently, to ulcerations of the intestine.

As was already stated, many cases run their course painlessly. When copious, the exudate is easily detected by the usual physical signs. In others it is encysted because of plastic fibrinous formation. Thor-mayer<sup>1</sup> described physical signs which he considers characteristic of tuberculous and carcinomatous peritonitis. He found that tympany is very frequently elicited on the right side of the abdomen, while in the left side a dull note is elicited by percussion. He explains this phenomenon on anatomical grounds: The mesentery on the right side usually contracts more than on the left, and thus intestinal coils are apt to be drawn to the right by the shrinking mesentery; tympany is then elicited over these distended intestinal coils. It is, however, an inconstant symptom and if it occurs at all, it is discerned late, after the organization of the exudate.

At times we may, on palpating the abdomen, feel some crepitation, and in some cases I have even heard friction sounds while auscultating with the stethoscope. On rare occasions, tumor-like masses are palpable in the abdomen. When localized in the right side they may simulate appendicitis. In one case under my care repeated attacks of pain in the right lower part of the abdomen, constipation, and even rigidity of the rectus muscle exquisitely simulated appendicitis. But later when a tumor was palpable the condition was cleared up. In another case under my care symptoms not unlike those of intestinal obstruction were present in a woman with tuberculous pleurisy, and the advisability of operative interference was seriously considered, but the patient recovered. It appears that tuberculous cicatrices causing narrowing of the gut may stretch and thus relief ensues. This is also true of cicatrices of the intestinal wall caused by healing tuberculous ulcers.

<sup>1</sup> Ztschr. f. klin. Med., 1884, vii, 378.

**Tuberculous Meningitis.**—Many phthisical patients show cerebral symptoms a few days before death, but at the autopsy no changes are found within the cranium. But in these cases the diagnosis is not important because the seriousness of the condition is evident from the other symptoms. The problem of the presence or absence of meningeal implication in phthisis has, however, a great prognostic value in cases showing a tendency to quiescence or cure, and the occurrence of symptoms suggestive of tuberculous meningitis is more than disquieting.

The onset of this complication is usually insidious. For some days, at times for more than two weeks, the patient complains of headache, is irritable and fretful and vomits most of the food and drink given him. Tuberculous patients only rarely suffer from headache, unless pyrexia, or some nasal or gastro-intestinal trouble is responsible. If a persistent headache cannot be explained as due to some other cause, meningitis is to be thought of. If there is also vomiting the diagnosis is greatly supported, though not conclusive. There are also noted early confusion of ideas, impaired memory, photophobia, defective vision, drowsiness and somnolence which may pass into coma, or convulsions.

The pulse is rather slow in most cases, though at times we meet with a case in which it is accelerated. But it is very frequently irregular. The temperature may be high, though this is rare. In most cases it does not exceed 102° F. Constipation is a frequent symptom, and during the last days retention of urine may occur. But these are not constant symptoms. Patients with diarrhea may continue with loose stools and in the later stages involuntary evacuation of urine and stools may occur.

In most of my cases many of these symptoms were noted early but they were not continuous; occurring one day and disappearing the next, to reappear again. This intermittency is a very important point in the diagnosis of obscure cases. Very early there is often noted a complete change in the character of the individual. The hopefulness and euphoria disappear: the patient becomes disinterested in things which were vital to him before. This passes into drowsiness and he refuses to answer questions, though when waked up he recognizes the person addressing him. Some act as if they were under the influence of alcohol, and in one case we suspected that the patient had imbibed whisky, and rebuked him for violating the hospital rules. Occasionally hysteria will simulate meningitis exquisitely. Kernig's sign is present in most cases, though in some it is lacking at the early stage. At the end Cheyne-Stoke's breathing, paralysis of some cranial nerves, optic neuritis and convulsions may occur.

In most of the cases under my care lumbar puncture has not been of material assistance for early diagnosis. Very often the fluid is cloudy, shows an excess of lymphocytes and is rarely sanguineous. But it must be mentioned that an excessive number of lymphocytes is not always a sure sign of tuberculous meningitis. In a large proportion

of cases the cerebrospinal fluid shows no change in its cytology, though the course of the disease and the autopsy leave no doubt that there was meningitis. In some, though not in all, tubercle bacilli may be discovered in the cerebrospinal fluid. Usually the fluid is under high pressure, but I have seen cases in which it squirted out forcibly, yet the subsequent course showed that there was no meningitis.

Patients with this complication do not last over two weeks, as a rule, though I have seen some who have lasted more than a month. A fatal prognosis should be given whenever meningitis is diagnosed; the few cases of recovery which have been reported may be considered medical curiosities.

**Cardiac Complications.**—We have shown that phthisis only exceptionally develops in persons suffering from chronic endocarditis, excepting in those with congenital heart disease (p. 91). But endocarditis may develop during the course of phthisis, either due to complicating rheumatic disease, or any other accidental septic process, as tonsillitis, etc. The verrucose excrescences on the cardiac valves often found at autopsies on phthisical subjects are usually caused by other microorganisms, though Heller, Leyden, Benda, Tripier, and others maintain that tubercle bacilli may be responsible in some cases.

**Myocarditis.**—In most cases heart failure in advanced phthisis is due to myocarditis, with dilatation of the right heart; to tuberculous pericarditis, and also to dilatation with cardiac displacement. Like in other chronic, cachectic, and exhausting diseases, the myocardium partakes in the atrophy of the muscular system, and gives way from sheer exhaustion. In fibroid phthisis, and the pleural forms of chronic phthisis, the induration in the lungs interferes with the circulation, and heart failure of variable degree is the result. Before the onset of decompensation, hypertrophy of the right ventricle is quite common, especially in fibroid phthisis.

Pulsations in the epigastrium and accentuation of the second pulmonic sound reveal this condition. However, accentuation of the second pulmonic sound may be present without hypertrophy when the left lung is retracted through infiltration or shrinkage and reveals the left heart. The constitutional symptoms of heart failure—dyspnea, edema, etc.—may be quite marked.

**Pericarditis.**—Pericarditis may occur during the course of chronic phthisis. Several cases of primary-tuberculous pericarditis have been reported. In chronic phthisis the pericardial sack may be implicated by tuberculous processes of the pleura or mediastinal glands. Adhesions between the pleura and pericardium are often found and with the shrinkage of the affected lung, the heart is pulled out of its normal position, as has already been described (pp. 335 and 344).

Very often we meet with acute pericarditis in phthisis and pleuro-pericardial friction sounds may be audible. The symptoms and signs of adhesive pericarditis are not rare in chronic phthisis—systolic retraction of the chest wall at the apex, engorgement of the veins in the

neck, disappearance of weakening of the pulse during inspiration—*pulsus paradoxus*, etc.

On very rare occasions we meet with acute pericarditis coming on suddenly with pain in the cardiac region, dyspnea, cyanosis, cardiac irregularity, etc. In one case under my care the symptoms simulated pneumothorax. Careful examination of the heart, however, clears up the case. The cardiac dulness is increased, friction sounds are audible, the apex beat disappears with the effusion. The pericardium may also be implicated in cases of pneumothorax, producing pneumopericarditis, as has already been mentioned.

**Phlebitis and Thrombosis.**—Although occurring quite frequently during the course of phthisis, phlebitis and thrombophlebitis are only rarely mentioned as complications of this disease. Older clinicians, as Hoffmann in 1740, and after him Hunter, Louis, Trousseau, and others have mentioned it, and Cursham wrote in 1860 on “Causes of Obstruction of the Veins of the Lower Extremities Causing Edema of the Corresponding Limb and Occurring in Phthical Patients.” Most writers are inclined to attribute them to the tuberculous toxemia, while others have found in them an instance of marantic thrombosis. But recently Gustav Liebermeister,<sup>1</sup> in a thorough clinical and pathological study of the subject, attributes them to the direct action of the bacilli on the bloodvessels, finding as he does that nearly all tuberculous patients have a bacteremia. Haushalter and Étienne, Vaquez, Sabrazés and Mongour, Chantemesse and Widal, Lesné and Revaut, Liebermeister, and others have found virulent tubercle bacilli in such thrombi.

Phlebitis and thrombosis in phthisis usually occur in the femoral vein, though at times we meet with cases in which the vena cava, the innominate, jugular, subclavian, or renal veins are affected or even the cerebral sinuses. The frequency of this complication is given by P. R. Dowdell<sup>2</sup> as 30 among 1300 consumptives, or 1.5 per cent. H. Ruge and Hierokles<sup>3</sup> found it nineteen times among 1778 cases of pulmonary tuberculosis, or 1 per cent. In my experience it appears to be even more frequent in advanced and active cases of phthisis. P. Horton-Smith Hartley found thrombosis of veins in 2.6 per cent. of 263 cases which came to autopsy. In males the percentage was but 1, while in females it was 6.6.

Phlebitis is very often found in the veins of the upper or lower extremities, especially in very active cases running high fever. Mostly the medium-sized or small veins are affected. Clinically, the thickening of the veins of the upper extremities are more easily recognized by palpation because of the lesser thickness of the muscles and adipose tissue. The affected veins are tender to the touch and also painful on motion of the limb. Edema of the extremities is exceptional in simple

<sup>1</sup> Virchows Archiv, 1909, exviii, 332.

<sup>2</sup> Amer. Jour. Med. Sci., 1893, cv, 641.

<sup>3</sup> Berl. klin. Wochenschr., 1899, xxxvi, 73.

phlebitis, though in some cases it may occur. The phlebitis may disappear, to reappear again and in most cases it is persistent till thrombosis also occurs, or till the fatal issue of the case. In fact, phlebitis is an ominous complication. A thrombus may develop and it may soften and be carried by the circulating blood to distant organs producing pulmonary embolism or infarction. It may organize and remain as a firm, thick cord. Hirtz<sup>1</sup> described cases of phlebitis and thrombosis occurring during the incipient stage of phthisis, or even preceding the actual onset of the disease, especially in chlorotic girls.

**Thrombosis of the Femoral Veins.**—Thrombosis occurs most frequently in the femoral vein but, as was pointed out by Dowdell, usually the popliteal vein is found to contain a clot of older date, while in some the saphena vein is plugged and rarely the superficial veins of the leg and thigh, as well as the main trunk from the tibial vein upward is thrombosed. Dowdell, Ruge and Hierokles, Liebermeister, and others have also found thrombosis of the uterine and brachial veins, the prostatic plexus, and embolism of distant arteries is said to be not uncommon. As is the case with phlebitis, thrombosis is found mostly in far-advanced but acutely running cases and is usually the precursor of a fatal issue.

The most important symptom is edema of the affected limb. The onset is usually slow and insidious, the swelling coming on gradually. Pain is often felt for a few days after the onset of edema, but in many cases this is lacking. When present it is mainly felt in the popliteal space where tenderness may be elicited. Inasmuch as practically all these patients have symptoms of active phthisis, the temperature is not an aid in the diagnosis—it is continuous or hectic, as the case may be; the onset of the thrombosis, edema, etc., has hardly any influence on the pyrexia. In some cases under my care there were disturbances in sensation of the affected limb, which was cold, numb, or tender. In one case the pain was excruciating and morphin alone was effective in relieving it in part. When the deeper veins of the muscles are plugged, which is not rare, there may be severe pain and hyperesthesia of the calf of the affected leg. Diagnosis may be difficult at first, but as soon as the edema appears, the cause is clear. In some cases the thrombus in the affected vein is so thick as to be palpable. I have many times been able to palpate the femoral and crural veins as thick, firm cords tender to the touch.

*Diagnosis.*—In most cases the diagnosis of thrombosis and phlebitis is rather easy. It is to be differentiated from edema of the extremities common in phthisis and due to cardiac and renal insufficiency, and from cachectic edema which is frequently seen in the terminal stages of this disease. Thrombosis always begins in one extremity and is confined to it, or marked on one side when fully developed. It is tender to the touch along the course of the veins and not necessarily

<sup>1</sup> Sem. Méd., 1894, xiv, 274.

over the edematous skin. The dilated superficial veins may at times contain clots. On the other hand, edema due to cardiac or renal disease is accompanied by signs and symptoms of these conditions, both lower extremities are affected by the swelling, and the tenderness along the course of the veins is lacking. Cachectic edema occurs on both sides, is painless and subsides when the patient is kept in the recumbent position for some time. At times intra-abdominal pressure on the common or external iliac vein, or on the femoral may produce edema of one extremity not unlike that of thrombosis. The same condition may occur, though very rarely, in the upper extremity when intrathoracic pressure is exerted by enlarged glands in the thorax on the main trunks of the veins. But careful examination will usually reveal the tumor or the glands which are responsible.

**Thrombosis of the Jugular Vein.**—Thrombosis causing edema of the upper extremity is very rare, but it does occur. Two cases have come under my observation. Humphrey<sup>1</sup> reported such a case in 1859; Lésague<sup>2</sup> observed in 1870 a case of phthisis complicated by the formation of a thrombus in the external jugular, subclavian, and humeral veins. Ten days after the appearance of the thrombus it was completely softened and all symptoms of phlebitis disappeared. But in all other cases reported, death supervened within a couple of weeks after the establishment of thrombosis. The symptoms are edema, pain, etc., of the upper extremity. In 1904 Charles J. Aldrich<sup>3</sup> collected from the literature 9 cases of this complication of phthisis and reported one of thrombosis of the left internal jugular with extension through the subclavian down the axillary into the basilic veins. Two weeks later a like thrombus appeared in the right side and extended to the veins of the arm. Death was due to cerebral sinus thrombosis from extension of the thrombus in the right internal jugular vein. In one of my cases thrombosis of the right internal jugular vein occurred in a patient with a spontaneous pneumothorax.

*Prognosis of Thrombosis.*—The prognosis is fatal in nearly all cases because of the severity of the tuberculous process, occurring as it does mainly in rapidly advancing cases of phthisis. Death may be due to secondary emboli which cause sudden death. Excepting Lésague's case mentioned above, I have not heard of a patient with phthisis complicated by thrombosis of the upper or lower extremity surviving two months; they usually succumb within one month.

**Urogenital Tract.**—Of other complications occurring more or less often during the course of phthisis may be mentioned tuberculosis of the urogenital tract. We have already mentioned that albuminuria is not uncommon in phthisis. In far advanced cases, nephritis is quite frequent and we may have most of the symptoms of this disease, especially edema, anasarca, etc., and even uremia, which

<sup>1</sup> Brit. Med. Jour., 1859, 582, 601, 619, 650.

<sup>2</sup> Gaz. Méd. de Paris, 1879, i, 649.

<sup>3</sup> New York Med. Jour., 1904, lxxix, 442.



is at times difficult to differentiate from tuberculous meningitis. In many of advanced cases we may also note symptoms due to amyloid disease of the kidneys: Abundance of secretion of urine of low specific gravity containing hyaline casts and albumin in large quantities. But in this form of nephritis dropsy is infrequent. I have been struck with the fact that in most cases in which there is considerable albumin in the urine and dropsy, the temperature drops down to near normal and very often the activity of the process in the lung diminishes. The prognosis is, however, not improved.

In some cases tuberculosis of the kidneys supervenes and also of the bladder, seminal vesicles, vas deferens and epididymis. Tuberculosis of the kidneys is very difficult of diagnosis in its early stages. Finding acid-fast bacilli in the sediment of the urine is not sufficient to base a diagnosis in my experience, excepting when the specimen has been obtained by catheterization of the ureter. But I have seen cases in which tubercle bacilli were thus found yet the patient improved without operation. Patients with tuberculous pyelitis suffer usually from lumbar pain of a dull character, have pus, albumin and blood, renal epithelium, and even caseous débris in the urine. I have seen cases in which the pain occurred in paroxysms and it was difficult to differentiate from that of renal colic due to stone.

**Tuberculous Ulcerations of Mucous Membranes.**—We have already pointed out that despite the fact that so much of tuberculous sputum passes through the mouths and lips of phthisical subjects, ulcerations of these parts are extremely rare. Still now and then we meet with cases of ulceration of the tongue. These ulcers are usually located on the dorsum of the tongue, but in some also at the sides, the tip, and rarely on the frenum. I have seen some with ulcers of the soft palate and very rarely on the posterior wall of the pharynx. These ulcerations are to be differentiated from specific and malignant ulcerations. Inasmuch as they occur usually in the far-advanced stages of the disease, the diagnosis is clear, but when occurring in a patient with a slight pulmonary lesion, or in one with the emphysematous type of fibroid phthisis the diagnosis may be difficult, though the examination of a specimen from the ulcer for tubercle bacilli usually decides.

**Purpura.**—I have seen several cases of purpura hemorrhagica complicating advanced phthisis. Petechia are very frequent in many cases, but true purpura hemorrhagica with extensive ecchymoses scattered over the limbs may occur, and there may be simultaneously hemorrhages from some of the mucous membranes—true purpura hemorrhagica. In 3 out of the 4 cases seen by me recently there was also albuminuria and hematuria, and the patients succumbed shortly after the appearance of the purpura, and I am inclined to agree with John M. Cruice<sup>1</sup> to the effect that the occurrence of purpura, espe-

<sup>1</sup> Amer. Jour. Med. Sci., 1912, exliv, 875.

cially the hemorrhagic form, in the course of tuberculosis is always a grave symptom.

Its etiological relation to tuberculosis is doubtful. Some authors are inclined to see in the tubercle bacillus a cause of the purpura, but the fact that it is so extremely rare in phthisical subjects shows that when the two diseases occur in the same subject, it is in all probabilities a coincidence. I believe that Cruice's observation that after an attack of purpura physical examination will reveal a more advanced condition of the lesion, does not at all prove that the hemorrhages into the skin were directly of a tuberculous character; it by no means excludes the chances of their being a coincidence.

**Superficial Cold Abscesses in the Chest Wall.**—This is a very rare complication of phthisis and may occur at any stage of the disease, mostly in the early or second stages in cases running a chronic course. The cause usually cannot be ascertained—they are often found unexpectedly. On the chest wall, along the line of insertion of the diaphragm, particularly anteriorly or in the lower axillary region there is noted a circumscribed swelling, the size of a pigeon's or hen's egg, painless and fluctuating. There is usually no surrounding inflammatory induration, and only later the infected area becomes red and somewhat tender. When incised a moderate amount of liquid, curdy pus is eliminated, but healing is slow: In most cases a fistula is left which persists for months; or an ulcer remains which keeps on discharging pus for a similar period. Very often the fistula or ulcer is located over a rib, the periosteum of which is implicated. In many cases healing finally takes place leaving an ugly red scar.

The diagnosis is at times difficult—there is a question whether it is not an empyema pointing on the chest wall, particularly when there are physical signs elicited in the same area. A careful consideration of the history and course of the trouble, however, clears up the diagnosis.

## CHAPTER XXVII.

### PROGNOSIS IN PULMONARY TUBERCULOSIS.

**The Curability of Phthisis.**—Laennec, the first physician to make a scientific study of the pathology of phthisis, pronounced it an incurable disease, saying "the possibility of curing phthisis in the first stage is an illusion." Pathological anatomy taught him that tuberculosis is an affection akin to cancer and absolutely incurable; that all tuberculous lesions proceed from infiltration to caseation and finally to softening. It appears, however, that even in those days it was already well known that if taken in time the disease is curable.

The observations of physicians all through the nineteenth century have clearly shown that phthisis is not invariably fatal, despite the fact that the treatment applied during the first half of the nineteenth century should have killed most of the curable cases, according to our understanding of the pathology and therapy of the disease. Still, Flint reported 670 cases observed during a period of thirty-four years and the proportion of cases cured or arrested was not much below that which we attain at present. Thomas J. Mays<sup>1</sup> compiled statistics of Flint's 670 cases and Williams's 1000 cases observed for twenty-two years, and compared the results with Trudeau's 1060 cases under observation for seventeen years. The percentages of recoveries and survivals are about the same, or rather in favor of Flint's and Williams's cases.

At present we have sufficient and uncontrovertible proof that tuberculosis is curable in all its stages. Experience while making autopsies shows, in fact, that it is the most curable of chronic diseases, considering the enormous number of persons who show healed or quiescent tuberculous lesions in the lungs when examined after death. And the lesions discovered are often such as to indicate that the process was quite extensive at the time of its activity.

**Importance of Prognosis.**—There is no need of elaborating on the importance of prognosis in the practice of medicine. It is always important and, in the case of tuberculosis, it is at times even more important than diagnosis. Indeed, most patients come with ready-made diagnoses and all they want to know is the ultimate outlook. "Will he recover?" is one of the first questions after the patient and his friends are told that there is a tuberculous lesion. "If so, how long will it take till he recovers?" Moreover, it is important to be ready to answer whether the patient after recovery will be able to resume

<sup>1</sup> New York Med. Jour., 1914, c, 70.

his occupation, and whether there is danger of relapse. In case of an unfavorable prognosis it is often asked "how long will the patient last?"

We cannot answer all or most of these questions in the average case with a high degree of certainty. As J. Mitchel Bruce<sup>1</sup> says: prognosis in tuberculosis "is always a difficult and often a disappointing proceeding. With all the facts of a case in our possession the conclusion we reach proves too frequently to be false. Indeed, paradoxical as it may appear, we fail in prognosis most often because of the very number, variety, and different character of the facts that we discover. Each of our observations has its own prognostic value, and most of them have a different value in different instances and at different times. We meet with an extraordinary, variable, and therefore uncertain, course of the pathological process from month to month. No disease is so difficult to deal with in this connection, and we have to confess that we too often find ourselves changing our forecast in both directions from time to time."

The difficulties are, however, not insurmountable in many cases, and we can estimate the prognosis of the average patient in any stage of the disease, with a certain degree of exactitude. But in order to do this, we must take into consideration all available facts which may have any bearing on the course of the disease.

**Elements of Prognosis in Phthisis.**—The notion that this disease is curable only in its incipient stage is one of the half-truths which have gained universal credence because of tradition. There are so many exceptions as to almost nullify this ancient dictum. We have already shown that it is fallacious to classify phthisis into three or four stages and to say without reservation that in the first stage it is curable; in the second stage the chances of recovery are considerably diminished while in the third stage it is incurable. There are "incipient" cases which have no chance, irrespective of the treatment applied; while there are many in the third stage whose chances of survival and even of efficiency are excellent. For this reason we shall not discuss the prognosis of phthisis according to the stages of the disease.

The elements of prognosis in phthisis reside in the following factors: (1) The form of the disease; (2) in a given form of the disease, the activity of the process as revealed by the constitutional symptoms and physical signs; (3) the presence of complications; (4) the extent of the lesion in the lungs; and (5) the economic condition of the patient.

**Prognosis in the Various Forms of Pulmonary Tuberculosis.**—We have seen from our study of the symptomatology of phthisis that the form of the disease has a greater influence on the ultimate outlook than the extent of the lesion or even the activity of the process. Thus, in the pulmonary form of miliary tuberculosis, the chances of recovery

<sup>1</sup> Lancet, 1913, i, 591.

are *nil*. The patient will die irrespective of the treatment applied. In acute pneumonic phthisis the prognosis is very unfavorable, the only hope we may entertain is that the disease will take a turn to the better and pursue the course of chronic phthisis. This happens on rare occasions, but should not be expected in the average case. In fact, we may say that the prognosis is decidedly bad in these cases. The patient usually lasts as many weeks or months as one with chronic phthisis lasts years.

On the other hand, taking the other extreme, abortive tuberculosis, we find that the prognosis is favorable under all circumstances. Practically all patients recover; the vast majority without even knowing that they have been tuberculous; or when the disease has been diagnosed there often remains a lurking suspicion that it was a false alarm, even if tubercle bacilli were discovered in the sputum.

In fibroid phthisis the prognosis is very good indeed, as long as there is no fever. The dyspnea and discomfort which this disease causes for years are bearable by the average patient. But as soon as fever makes its appearance, and persists for some time, the prognosis is that of chronic phthisis which will soon be discussed.

The influence of the patient's age on prognosis has already been discussed in the chapters dealing with tuberculosis in children (p. 374 and 391), and in the aged (p. 394).

The most important form of phthisis, that of the most common chronic type, is the disease in which the prognosis is very difficult to formulate in the individual case. We may be able to prove statistically that a certain percentage of cases recover completely; another percentage will survive so many years; still another percentage will succumb within one or two years, etc. But in the practice of medicine we deal with individual cases and statistics count for naught.

In the individual case the outcome of the disease depends on so many complex and variable factors, that it is often very difficult to formulate a prognosis. Indeed, we see that the most desperate case, slowly or suddenly, with or without any discoverable reason, takes a turn to the better and recovers. We see others who drag along for years, living, but they do not recover. Still others, in whom the general condition has been quite or altogether favorable, suddenly take a turn to the worse and the patient is carried off within a few weeks or months.

For these reasons we must enter into the elements of prognosis of chronic phthisis in greater detail.

**Prognostic Significance of the Patient's History.**—Many authors have stated that patients with a family history of tuberculosis are more likely to run an unfavorable course than those derived from non-phthisical stock. A consideration of the facts brought together in Chapter V will show that this is a fallacious view. The patient was undoubtedly infected during childhood. Had he suffered a massive infection during infancy he would have succumbed to some acute

form of tuberculosis. The fact that he survived the primary infection proves that it was mild; this is also the reason why he now suffers from chronic phthisis, and not from an acute form of the disease. Indeed, patients showing signs of some local tuberculous lesion at an earlier age usually have a slow, sluggish form of phthisis lasting for many years. Many authors have also calculated that the average duration of a phthisical patient with a family history of tuberculosis is longer than in one derived from robust stock. This is best seen in the acuteness of phthisis in persons who have just emigrated from rural districts into large cities.

Experience teaches that the prognosis is not different in adults who are derived from phthisical stock than in those who are not. The slight differences that have been discerned appear to be rather in favor of the former.

**Sex.**—It appears that the prognosis is more favorable in women than in men. A man acquiring tuberculosis is apt to continue working and thus aggravate the prognosis while a woman, who is usually not the bread-winner, is more likely to abstain from overexertion, which is such an important element in the treatment of this disease. On the other hand, pregnancies, labor, and lactations are apt to aggravate the prognosis in women. In fact, it has been my experience that the prognosis of phthisis in women is better in those who are unmarried than in those who are married. Women are less likely to succumb to some of the more serious complications of phthisis, such as hemorrhage, pneumothorax, etc. They also less often suffer from laryngeal tuberculosis.

**The Onset of the Disease.**—In cases with a sudden onset the prognosis is worse than in those in whom the disease came on insidiously. Even the fact that the former are more apt to take strong measures to prevent the activity of the process does not counterbalance the seriousness of an acute onset, excepting when the suddenness refers merely to an initial pulmonary hemorrhage. An acute onset means severe constitutional and toxic symptoms, low powers of resistance, and the process in the lungs extends very quickly, so that in a short time quite large portions of one or both lungs are affected.

Those beginning with hemoptysis have usually a better outlook than others. The reason is not clear. Perhaps the dramatic onset frightens the patient and he is apt to institute proper treatment even if he feels well after the cessation of the bleeding, while patients with mild symptoms but without hemoptysis may continue at work till the disease is aggravated. But this does not explain all cases. It seems that hemoptysis has very often a good influence on the prognosis of phthisis at any stage of the disease and many patients feel much better after a brisk hemorrhage (see p. 201). The cases marked by an onset with pleurisy, dry or moist, have, as a rule, a better prognosis than others, as has already been stated (p. 89). It has been observed that patients who are only slowly regaining their health after

an attack of pleurisy, are pale and emaciated, and are more likely to develop active and progressive phthisis than those who recover quickly and soon regain their former health.

**Prognostic Significance of the Activity of the Disease.**—We have seen throughout this book that the activity of the process in the lung has a greater influence on the ultimate outcome than the stage of the disease. The activity is best studied by a careful consideration of general or constitutional symptoms. Of these, fever is the most important. There is no active tuberculosis without pyrexia. The afebrile cases discussed elsewhere are rather uncommon and it is a fact that the prognosis is rather good, as long as fever is lacking. Each turn for the worse, each complication, is accompanied by a rise in the temperature.

In active disease the prognosis is unfavorable in direct ratio to the height and duration of the fever. Every extension of the lesion manifests itself by increased pyrexia; persistence of pyrexia, despite rigid rest in bed, is pathognomonic of low resistance; the reverse type of fever, in which the highest point is reached in the morning instead of the afternoon or evening, is of grave prognostic significance—it may be an indication of an invasion of both lungs by tubercles. On the other hand, moderate fever, less than 101° F. dropping down to normal or subnormal in the morning, is rather favorable. In other words: The higher the morning temperature, the nearer it approaches the evening temperature, the worse the prognosis. Hectic fever, with normal and subnormal temperature in the morning, but which rises high in the afternoon and evening, is of grave prognostic significance. If it lasts for more than a month, the patient will not survive. He may last or even improve for a time, but he will not recover.

A normal temperature throughout the day and night is a good sign; when accompanied by a good appetite, gain in weight, diminution in the cough and expectoration, etc., it is an indication of healing of the lesion. If fever only ensues after exertion or excitement, the prognosis is very good indeed, provided proper treatment is instituted. It is for this reason that most who have new and infallible remedies for phthisis ask for just this sort of cases on which to try the treatment. The vast majority recover under any treatment, provided good nourishment and rest is part of the proceeding.

Indeed we can, in most cases, formulate our prognosis by a careful study of the temperature curve for a few weeks. Of course we may on rare occasions err by putting implicit faith in the temperature curve, but the proportion of errors will be less than when we attempt to formulate it on other data, especially on the stage of the disease, or the findings on physical examination.

For this reason, *a prognosis in phthisis should not be given after a single examination of the patient.* It is required that the temperature of the patient should be studied for at least two weeks before attempting to forecast the outlook.

The prognostic significance of the pulse should be considered. Excepting in heart disease and hyperthyroidism, no disease can be evaluated prognostically with the same degree of accuracy by the pulse rate as chronic phthisis. Incipient cases with a pulse not above 80 per minute have an excellent outlook. Tachycardia is an indication of acuteness of the process, or low resistance, or both. Patients who have apparently recovered but remained with a rapid pulse, have a very poor outlook. The outlook is good in chronic cases with slow pulse.

Of the other constitutional symptoms which give us prognostic hints, the state of the gastro-intestinal tract is of great importance. Patients with good appetite and who digest and assimilate their food well, recover, even when they have, for the time being, some fever every afternoon. Persistent anorexia and gastro-intestinal disturbances are of grave prognostic significance. Gain in weight in afebrile patients with good appetites is a good sign. But occasionally we meet a patient who holds his own or even gains despite the fever. In such cases the thermometer should be our guide, and not the scale.

Hemoptysis has no influence on the course and prognosis of the disease in the vast majority of cases. The initial hemoptyses are rather salutary, as was stated above. No patient has succumbed to a really initial hemoptysis. Ninety-eight per cent. of cases of advanced disease recover from hemorrhages. But in cavitory cases, which may or may not be doing well, a brisk hemoptysis may unexpectedly kill the patient. If in the average case the hemoptysis is not accompanied by fever, or the fever lasts only a few days after the cessation of active bleeding, the prognosis is good. But if pyrexia continues it may point to acute pneumonic phthisis or tuberculous broncho-pneumonia which is almost invariably fatal. In these cases the hemoptysis is indirectly responsible for the fatal issue.

**Prognostic Significance of Complications.**—The presence of complications, tuberculous and others, modifies the prognosis perceptibly. Thus, laryngeal and intestinal tuberculosis aggravate the prognosis. Though many recoveries are seen in patients with these affections, yet in the individual case we must not give a favorable prognosis in those who show positive proof of laryngeal or intestinal complication. With advanced laryngeal disease, manifesting itself in aphonia, dysphagia, etc., a fatal issue is to be expected. The same is true of diarrhea which lasts more than a month. We occasionally, however, see patients with profuse diarrhea lasting for several months. But they never recover. Blood in the stools is another unfavorable sign. Ischiorectal abscess is itself an indication of intestinal tuberculous ulceration and is of unfavorable prognostic significance.

Pleurisy is not invariably an unfavorable complication. The dry form occurs in nearly all chronic cases and has a rather salutary influence on the pulmonary lesion; it is also a good preventive of spontaneous pneumothorax. Pleural effusions are serious, though in many



cases they have a good influence on the basic disease. We have already shown that they occasionally promote the healing of the lesion in the lung by compression. But in bilateral lesions the side with a free pleura is likely to suffer from an extension of the tuberculous process and the outlook is gloomy.

Empyema is a very bad complication. No recovery is to be expected. The patient may last for months, but he will not recover. On exceedingly rare occasions the pus breaks through a bronchus and is expectorated. But even here the ultimate outlook is bad, because of the amyloid degeneration of the viscera and the general malnutrition caused by the prolonged suppuration.

Spontaneous pneumothorax is fatal in 95 per cent. of cases within one month of its occurrence. The exceptions have already been mentioned.

Tuberculosis of the kidney is of unfavorable import.

Of non-tuberculous complications we may mention influenza. This disease is more often diagnosed in tuberculous patients than facts would warrant. An increase in the cough, pyrexia, etc., due to an exacerbation of the tuberculous process, is apt to be attributed to influenza by patients and physicians. But when it does occur, it is apt to wake up quiescent lesions in the lungs. Lobar pneumonia occasionally occurs in phthisical patients. In the cases observed by the author the outcome depended on the condition of the tuberculous lung. Those with slight quiescent lesions may pass through an attack of pneumonia, recover, and the phthisis should pursue its course as if no such complication had occurred. But in patients with extensive tuberculous lesions, reduced in vitality, the pneumonia is the last straw and the patient is carried off within a week.

We often meet other non-tuberculous diseases in patients suffering from phthisis. Such as necessitate an operation with the administration of a general anesthetic are dangerous, and it has been my rule to urge local anesthesia whenever feasible in operations on tuberculous subjects.

Pregnancy is a grave complication of phthisis, and in incipient cases it is advisable to induce abortion whenever it occurs. For this reason it is urgent that married phthisical women should be instructed in the methods of prevention of conception. During pregnancy the patient may feel well, even better than before conception has taken place. But after childbirth there is often a reactivation of the tuberculous process and an acute course of the disease.

**Prognostic Significance of Signs found on Physical Examination.**—We have already mentioned the fallacy of formulating the prognosis of phthisis solely on the findings by physical examination. There are cases showing physical signs indicating that we are dealing with incipient, or first-stage cases of the American or Turban's classification, yet the prognosis is very unfavorable. Indeed the most unfavorable prognosis should be given in cases showing marked constitutional

symptoms which are out of proportion to the findings on physical examination.

It may be stated that generally the extent of pulmonary involvement is of more importance than the stage to which the lesion has advanced. Cavitation in one lobe is of less danger than infiltration of two or three lobes. J. Edward Squire gives the following table embracing 2720 cases of phthisis showing the relation of improvement to the number of lobes involved:

Lobes affected.	Cases.	Much improved. Per cent.	Improved. Per cent.	Total improved. Per cent.
1 . . . .	877	58.38	28.62	87.00
2 . . . .	1015	37.83	34.67	72.50
3 . . . .	515	22.52	35.53	58.03
4 . . . .	277	15.16	29.24	44.40

The fear and apprehension entertained by both the profession and the patient for "holes in the lung" are based on misconceptions of the pathology of phthisis. The fact is that the most dangerous cases of progressive phthisis are fatal before cavities are formed. This is the case with miliary tuberculosis and, to a certain extent, with acute pneumonic phthisis. If a tuberculous lesion in the lung does not cicatrize quickly, the best that can happen to the patient is that a cavity should form. A cavity is proof that the organism is in possession of strong powers of resistance, in fact of immunity; otherwise the lesion would spread. The difference between active phthisis with cavity formation and without such occurrence is analogous to that between general septicemia and abscess. In the latter case the disease is localized and circumscribed and, when drained, the danger is not very great. A cavity has, in fact, been defined as a tuberculous abscess which is drained through a fistulous opening into a bronchus.

It may be stated that the dangers of tuberculous cavities vary inversely with the time it takes for their formation. The sooner they are produced the worse the prognosis; the slower they develop, the better the ultimate outlook. In very acute forms of phthisis cavitation is very rare. The prognosis is gloomy with or without localized destruction of pulmonary tissue. In adults, such cases are comparatively rare, but in infants rapid cavity formation is seen at times and the termination is almost invariably fatal. In subacute forms of phthisis, in which excavations are apt to form very rapidly, the prognosis is unfavorable, unless the cavity is rather small. In the latter case the disease may be attenuated and subsequently pursue a chronic course with the sequestration and expulsion of the affected area. Excavation is then the first step toward the diminution of the acuteness of the process in the lung. The general symptoms may be ameliorated, as after the evacuation of an abscess.

In chronic phthisis excavations, even when extensive, are compatible with a long and efficient life. These cavities are surrounded by more or less dense fibrous capsules which limit their extension and

are drained through fistulous tracts communicating with bronchi. As long as the secretions are eliminated by expectoration, the patient may feel quite comfortable for years. The cavities may even heal, as was already shown (see p. 137). When small, they may be obliterated by granulations or by calcification of their contents. Larger excavations may shrink or, even when remaining of large dimensions, they may become altogether benign after the necrotic tissue has been expelled. They are, however, a constant source of danger for metastatic auto-infection or copious hemorrhages.

In my experience patients with right-sided lesions of this type are more likely to recover than those with left-sided lesions. In the former the constitutional symptoms, especially dyspnea, tachycardia, etc., may improve or disappear after the formation of a chronic cavity and the disappearance of the pyrexia. Even dextrocardia may be well borne. But in left-sided lesions the heart is pulled over to the left and upward, and the patient remains with tachycardia and is distressingly short-winded. Though he may last for years, he never regains efficiency.

In chronic cases in which the formation of a cavity is slow, the prognosis is rather good. In fact, cavity formation, as we have already shown, is a sign of immunity. Those with little or no resistance succumb before there is an opportunity for cavity formation.

These cavities are surrounded by dense fibrous capsules which limit their progress or extension, and they may be harmless for long periods of years. Communicating with bronchi which permit the expulsion of the morbid secretions forming on the ulcerated wall, they often pursue an apyretic course. Some even have smooth and glittering walls without any lymph spaces and the toxic products within them cannot be absorbed. We meet with cases in which even the tubercle bacilli disappear from the sputum and the prognosis is the same as in bronchiectasis.

There are many of this class of patients who, despite having more or less extensive excavations, live for many years without pronounced inconvenience; in fact some consider themselves fairly healthy and attend to their callings or even to manual labor. Their main trouble consists in a proclivity to "catch cold," and only on such occasions do they call on their physicians for relief.

Generally speaking tuberculous cavities are indications of chronicity of the tuberculous process in the lung, showing that the resisting forces are active and as such are of better prognostic augury than many active incipient cases.

Patients are to be told that the "holes" in their lungs *per se* are not as dangerous as they believe. That fever, anorexia, etc., are more dangerous. They may live and can be active with cavities for many years.

**Special Tests.**—Various attempts have been made to find tests of the severity of phthisis by examination of the blood, urine, etc. We

have already seen that Arneth's blood picture is not as reliable as some would lead us to believe (see p. 225). Ehrlich's diazo-reaction was at one time considered reliable in indicating the severity of phthisis. But it appears that it is positive in cases which are otherwise indicating their progressive tendencies. In incipient cases it is as a rule negative, but I have met with cases in which it was positive, yet the case went on to uneventful recovery. It appears that at present very few place great reliance on this test.

Moritz Weisz<sup>1</sup> found that urochromogen is the principal substance which causes the diazo-reaction and suggested that his test is superior to the latter. I have used of late Weisz's urochromogen test and found it superior to the diazo-reaction in indicating the prognosis of active phthisis. It is thus performed: Into each of two small test tubes are put 8 c.c. of urine and 2 c.c. of distilled water are added; now, to one tube which is to be tested for urochromogen, 3 drops of 1 to 1000 solution of potassium permanganate are added, the tube is shaken thoroughly and compared with the control tube. The appearance of the faintest yellow color shows the presence of urochromogen and is easily detected by comparing with the control tube, to which no potassium permanganate is added. The test is read positive, however, only when the solution stays clear.

In this country Heflebower,<sup>2</sup> and J. Metzger and S. H. Watson<sup>3</sup> have reported that this test is a reliable guide in estimating the activity of the tuberculous process and gives indication as to prognosis. I find that it is positive during acute exacerbations of the disease and is usually negative in incipient cases or even in quiescent cases. In acute progressive cases it is found positive and it becomes more and more intense with the extension of the disease. It is negative in most favorable cases.

The complement-fixation test, which has of late been used in the diagnosis of tuberculosis with doubtful results (see p. 324) has been found by some authors to have some prognostic value. Debains and Jupille<sup>4</sup> report that in active incipient and hopeful cases of phthisis the reaction is usually positive, while in advanced cases, with pronounced emaciation the reaction is often feeble or altogether negative. They try to explain these phenomena on the assumption that in progressive and advanced phthisis the antibodies in the serum have already been bound or neutralized by the substances produced by the tubercle bacilli. They also found that in experimental tuberculosis in rabbits complement-fixation activity goes hand-in-hand with the resistance of the animal. On the other hand, in tuberculous pleurisy with effusion negative reactions were mostly found, and this form of the disease cannot be considered as of especially unfavorable prognosis. Most of

<sup>1</sup> Münch. med. Wehnschr., 1911, lviii, 1348.

<sup>2</sup> Amer. Jour. Med. Sciences, 1912, cxliii, 221.

<sup>3</sup> Jour. Amer. Med. Assn., 1914, lxii, 1886.

<sup>4</sup> Compt. rend. Soc. de biol., 1914, lxxvi, 199.

the work along these lines was done by Besredka,<sup>1</sup> who in a recent paper reports that the reaction is uniformly positive in early cases of phthisis; in moderately advanced cases it is positive in the majority. With the advance of the disease the reaction becomes feeble, and finally in the terminal stages of phthisis it becomes negative. With Manoukhine he regards a negative reaction in advanced phthisis as a sign of approaching death.

These findings are worthy of further investigation because a prognostic test in phthisis is almost as important as a diagnostic test.

**Influence of Economic Conditions of the Patients on the Prognosis.**

—The occurrence of phthisis is in itself an indication of poverty. To be sure, we meet with numerous rich consumptives, but economic prosperity is not always an indication of rational life, proper food, regular hours, avoidance of physical and mental overexertion, etc. But in a given case of phthisis the prognosis is often influenced more by the social and economic condition of the patient than by any other single factor. After all, phthisis is the most expensive of diseases because it disables the patient for a long period of time and requires expensive treatment, including nourishment, a favorable home, etc.

The patients who can afford to bear the expense are more likely to recover than those who cannot. The artisan often has a family depending on him for support, and he is likely to keep at work while sick, till the disease has progressed to a stage where he can do no more and drops from sheer exhaustion. It is in these cases that the institutions, as well as the social service of modern enlightened communities do considerable to improve the prognosis of phthisis. But it must always be borne in mind that these agencies can do much better than merely give advice about the dangers of living with tuberculous persons in one home, and distribute sputum cups. If they do only this, the prognosis is often aggravated because the patient is at times treated like a pariah by his relatives and friends who are frightened by the numerous "visitors," the social workers, nurses, physicians, and others. I have seen families broken up in this manner; families in which there were no infants, and there was no reason to fear dissemination of the disease. But what is of most importance, the patient, deprived of the comfort of a good home, becomes despondent and the lesion progresses more quickly than it would otherwise.

**Antagonistic Diseases.**—We have already seen that individuals suffering from mitral stenosis are less likely to develop phthisis, despite the fact that they are just as much exposed to infection as others (p. 91). In fact, it appears that a hypertrophied heart due to any cause is more or less of a protection against phthisis; if the latter does occur, it runs a milder course and tends to heal.

Phthisis is characterized by arterial hypotension, and this may be the reason why it is so rare in patients with arteriosclerosis, and

<sup>1</sup> Ann. de l'Inst. Pasteur, 1914, xxviii, 569; Compt. rend. Soc. de biol., 1914, lxxxvi, 197.

when it does occur it runs a benign course. In fact, it is rare to find arteriosclerosis in phthisical patients with albuminuria, casts, etc., indicating that they have chronic nephritis. Similarly persons suffering from interstitial or parenchymatous nephritis of a chronic type become phthisical only rarely. In the aged—arteriosclerotics—phthisis runs an exceedingly chronic course, as we have already shown.

French authors have described an antagonism between the arthritic and the phthisical diatheses. M. Raynaud noted that in gouty individuals phthisis, when it does occur, has a better outlook than in the average patient. The lesion is usually limited to one apex and runs a latent course. A marked tendency to fibrosis is seen in and around the lung lesion. Well-nourished consumptives—the “fat consumptives” already mentioned—are mainly found among arthritic subjects, or persons of arthritic stock, and also among those who were scrofulous during early childhood, as has been shown by Pidoux,<sup>1</sup> Sokolowski,<sup>2</sup> and others. Even when they suffer from hemoptysis, which is not rare, they recuperate rather quickly and are none the worse for their experience. Lemoine<sup>3</sup> maintains that tuberculous arthritics supply the main contingent of the curable cases of phthisis, and among them are those who, despite tuberculosis, reach an advanced age. The nutrition of the patient is also affected to a lesser degree in scrofulous individuals when they become phthisical, even when the process is extensive. He believes that the tendency to evanescent congestive conditions promotes sclerosis of the lesion. But we now have a better explanation. Scrofulous individuals are endowed with a high degree of immunity against tuberculosis.

English writers, who have seen many gouty patients, confirm these observations. J. E. Pollock believed that “gout, like rheumatism, when the specific attack of the disease is developed in a case of tubercle, retards the latter.” Sir Dyce Duckworth supposes gout or the gouty diathesis is antagonistic to phthisis. F. Parkes Weber<sup>4</sup> suggests that the resistance of gouty persons toward tuberculosis is probably partly due to the meat food (butcher’s meat, eggs, and all animal protein foods) which most persons with acquired goutiness have been accustomed to indulge in freely during most of their lives. He suggested that there might be some substance circulating in the blood in gouty persons in minute quantities, yet sufficient to have an antagonistic action toward the growth of tubercle and that perhaps this was likewise the case in persons taking an unusual amount of food, which might partly account for the good results following the extra feeding of phthisical patients, when duly assisted by hygienic surroundings. “Great meat eaters, if not alcoholic, rarely, even in the

<sup>1</sup> *Études générales et pratiques sur la phthisie*, Paris, 1873.

<sup>2</sup> *Deut. Arch. f. klin. Med.*, xlvii, 558.

<sup>3</sup> *Semaine Médicale*, 1900, xx, 103.

<sup>4</sup> *Lancet*, 1904, i, 924.

most unhygienic surroundings, become phthisical." Sir Andrew Clark,<sup>1</sup> Herman Weber,<sup>2</sup> and others noted the antagonism between gout and phthisis. Weber even urges the acceptance as insurance risks of persons affected with fibroid phthisis, also such as have gout and tuberculosis, because they have great resistance against the ravages of phthisis. Bandelier and Röpke found that in individuals with a disturbed purin metabolism, phthisis is always chronic or latent and shows strong tendencies to fibrosis. Raw<sup>3</sup> regards the gouty diathesis as antagonistic to tuberculosis and he found that the blood of a gouty person is not a suitable medium in which the bacilli will flourish.

From personal experience the writer is inclined to agree with Mayer<sup>4</sup> that the antagonism applies only to constitutional gout, while gout resulting from plumbism rather favors the development of phthisis. I have, in fact, seen many cases of subacute phthisis, running a rapid course in house painters who have for years suffered from lead poisoning and atypical gout. Most of them, however, suffer from fibroid phthisis.

It also appears that syphilis, while not antagonistic to the development of phthisis, yet influences the latter disease so that it runs a mild course, showing strong tendencies to fibrosis. Fibroid phthisis is very often seen in old luetics, and antisyphilitic treatment has a good influence on both diseases. On the other hand, when a consumptive acquires syphilis both diseases are apt to run a rapid, or even a malignant course.

**Prognosis in Arrested Disease.**—We have seen that only lesions of abortive tuberculosis are completely healed by cicatrization and calcification. But this form of the disease is not recognized, as a rule, during its activity, and the prognosis is good at all events. It is different with chronic phthisis which has lasted for some time and finally there is an abatement in the constitutional symptoms and the patient is considered cured.

Cure by *restitutio ad integrum* is out of the question in these cases. The cicatrized and calcified foci usually contain virulent tubercle bacilli which may at any time become active again, flaring up the lesion, or causing metastatic auto-infection. Experience has taught that in the vast majority of cases these patients attain but "quiescence," and the term "arrested disease," which has recently been substituted for the term "cured," which was formerly in vogue, is proper. The patient is justified in asking for an opinion whether this arrested condition is likely to be lasting, or whether he will sooner or later suffer from a recrudescence of the symptoms of phthisis, a relapse, which is in fact an acute or subacute exacerbation. In other words, is the arrest of the disease an indication of a more or less permanent freedom

<sup>1</sup> Trans Med. Society, London, 1889, xiii, 9.

<sup>2</sup> Medical Examiner, 1898, p. 122.

<sup>3</sup> Tuberkulosis, 1911, x, 169.

<sup>4</sup> Ztschr. f. Tuberkulose, 1914, xxiii, 243.

from tuberculous sickness, or is it merely a long remission in the progress of the disease?

These problems can be solved by a consideration of the physical signs found in the chest, but with greater certainty when the constitutional symptoms are considered.

Physical exploration of the chest discloses usually signs of cicatrization of the involved lung tissue, pleural adhesions, evidences of fibrosis, while the rest of the lung may show indications of emphysema. Adventitious sounds are usually, though not invariably, absent; the case is "dry." Exquisite amphoric breath sounds may be heard over the site of cavities, combined with amphoric whispered voice but no rales. In others, the site of the lesion is only discovered by the dulness on percussion, and feeble breath sounds and sibilation are found over a circumscribed area of the chest, usually the upper part of one side. In many there are found signs of displacement of the mediastinum. But we have already emphasized the fact that the physical signs elicited on the chest are of but little value prognostically. The writer is under the impression that a patient showing a well-defined line of demarcation between the normal lung and the affected part, has a better prognosis than one showing a gradual change from normal to pathological lung tissue. But to this there are many exceptions.

The problems "Will the quiescence last?" and "Is the patient in danger of a relapse of the disease?" can best be answered by a careful consideration of the constitutional symptom. In general terms it may be stated that the patient is in danger of two accidents: (1) pulmonary hemorrhage; and (2) reactivation of the disease.

Pulmonary hemorrhage cannot be foreseen in these cases; nor can it be prevented. It may occur when the patient is in excellent condition. When not copious it merely frightens him, but even brisk and copious hemorrhages are well borne by 98 per cent. of patients; in fact they feel better in many cases after recovery from the bleeding, and quickly recuperate. Some have one such large hemorrhage a few years after recovery from the phthisis and feel well for many years thereafter or even for the rest of their natural lives. But in about 2 per cent. of these bleeders the hemorrhages prove fatal. As was already stated these hemorrhages cannot be foreseen nor prevented. Those suffering from "recurrent hemoptysis" hardly ever perish because of the bleeding. The danger is a brisk hemorrhage occurring suddenly in one who may not have bled before.

The constitutional symptoms are better guides in prognosis as to the chances of a lasting quiescent period. Most of these patients with arrested phthisis remain emaciated, anemic, with wasted muscles, often presenting a cadaverous appearance. Despite this, many of them are very active at their avocations and in fact they display energy and perseverance which is surprising when considered in connection with their physical decrepitude. Some are rather well nourished despite the fact that physical exploration shows a lesion of various



degrees of activity, from cicatrization to excavation. In my experience, patients apparently well nourished with quiescent or arrested lesions of this class are not as a rule doing as well as those of the lean type, despite their well-nourished bodies. We should not allow ourselves to be deceived in attempting a forecast by the amount of fat the patient has, by the fresh and browned skin which is often merely a superficial mask of improvement while the interior of the organism is vitally undermined.

The prognosis in these two classes of patients can only be determined with some degree of certainty by an analysis of the following conditions: If the improvement has been attained through careful treatment in a favorable environment, the test is whether the patient remains in good condition for some time after returning to his old environment without suffering a relapse of the constitutional symptoms. The test, in other words, is duration; improvement counts if it lasts without special treatment.

As long as there is but little cough or none at all, no fever, no tachycardia, dyspnea, chills, sweats, etc., the prognosis is good, no matter what physical exploration discloses. Continuous freedom from these symptoms for several months is an indication of arrest, even if tubercle bacilli are found in the sputum, while in those in whom arrest has just been attained, the prognosis is uncertain until time has shown that there is no tendency to recrudescence. The prognosis is even better in those who, despite resumption of their previous occupation, or taking up a new one, and living a rational, though not an exceptionally careful life, still keep in good condition. On the other hand, in those who purchased quiescence or arrest of the disease by special treatment, rest, and extreme care, the prognosis is less favorable, unless resumption of ordinary activities of life proves that recrudescence does not occur.

In short, the prognosis of quiescent and arrested disease can only be made by a careful observation for several months, and noting the effects of resumption of activities of life on the condition of the patient.

## CHAPTER XXVIII.

### THE INDICATIONS FOR TREATMENT OF PHTHISIS.

THE indications for treatment in pulmonary tuberculosis appear at first sight to be simple and clearly defined. On the principle that the first thing to do is to remove the cause, it would seem that there are but two procedures to follow: To destroy the bacilli which have settled within the body; or to increase the resisting powers of the patient, and thus render the soil unsuitable for the growth of the invading virus. But in this case the ideal, like other ideals, cannot be achieved in the average case, and the aim at curing the patient by the first of these procedures is not feasible at the present state of our knowledge.

We have no chemical remedy which will destroy the bacilli harbored within the body without simultaneously killing the patient. We have no drug which will render the tubercle bacilli harmless in the body, as quinin destroys the *Plasmodium malariae*, or salvarsan and mercury destroy the spirocheta in syphilis, leaving the patient in good shape. Even the so-called specific treatment—the various tuberculins, sera, and vaccins—which have been lauded for their alleged curative powers when properly administered, are not stated to have any known bactericidal action, nor are they known to hinder the proliferation of the bacilli within the body, or to immunize the tissues against the poisons engendered by these microorganisms through the production of antibodies, as is the case with antitoxins. Attempts at active immunizations have not met with notable success in tuberculosis.

The etiology of tuberculosis, however, teaches a lesson in rational therapeutics. The tubercle bacilli do not grow with equal facility in every individual; if they did, the number of human beings who succumb to this disease would be equivalent to the number that give positive reactions to tuberculin, indicating that they have been infected with tubercle bacilli—over 90 per cent. of the adult population in large urban centres. We have seen that the bacilli can proliferate and produce their noxious effects only in persons who offer a favorable soil for their existence.

In what this favorable soil consists, we are not altogether clear. In the chapter on Predisposition we discussed it in detail and it was evident that everything which undermines the general health of a person and reduces his vitality may prepare a favorable soil for the growth of tubercle bacilli within the body and thus produce phthisis. As a corollary we may argue that anything which will stimulate the vital defensive forces, which are more or less inherent in every indi-

vidual; or which will improve the nutrition of the body, may hinder the proliferation of the bacilli, and with the improvement in the general physical condition of the patient, the local lesion may cicatrize, or the dissemination of the bacilli by metastasis may be prevented.

This is what modern phthisiotherapy is aiming at in handling each individual case of the disease. As has been pointed out by G. Schröder,<sup>1</sup> modern therapeutic tendencies which are based on the achievements of immunology, have not changed our methods of treatment of tuberculosis, especially phthisis. It is today, as it was hitherto, based on the general principles of therapeutics, because phthisis cannot be considered an infectious disease *sui generis*. It can only originate in individuals with a certain constitutional susceptibility, which may be inherited or acquired.

**Air, Food, and Rest.**—The traditional therapeutic triad—air, food, rest—has withstood the test of time, and is at present called into service more often than ever before in the treatment of phthisis. Indeed, like many other excellent therapeutic agents which have become standard, it is very often abused. Many patients know of it and quite often tell their doctor that they are aware of the fact that medicine is helpless and that air, food, and rest is all that they need. Curious to say, some physicians do not protest.

But this is all wrong. The medical man of today has many more resources in his attempts at curing phthisis, and should not rely on the above-mentioned triad exclusively. Indeed, a physician who advises a patient to lead an open-air life in some region famous for its beneficial effects on this disease, and urges him to consume more and better nourishment than he has been in the habit of taking, and to stop all life activities, fulfills but part of his duty to his patient. There are many more therapeutic resources which hasten recovery, relieve the most annoying and painful symptoms of the disease, and go a long way toward prevention of complications, which cannot be met by the above-mentioned indications.

**Effects of Polymorphism of the Disease on Therapeutic Indications.**—Since the etiological unity of tuberculosis has been proved by the discovery of the tubercle bacillus, the profession has tacitly accepted that unity of origin invariably implies unity of effect, and the treatment of the disease was also unified. But this is an error. We have seen that the tubercle bacilli produce different lesions in different individuals, as regards the anatomical changes in the lung, the clinical phenomena and the course and curability of the disease. Indeed, there are hardly two cases of phthisis which appear exactly alike on the autopsy table, and all the groupings into caseous, fibroid, cavitary, pneumonic, etc., are inadequate. This is especially true of the clinical manifestations of the disease; its polymorphism is noteworthy and important. To be sure, this is also true of other diseases, notably

<sup>1</sup> Handbuch der Tuberkulose, 1914, ii, 1.

syphilis, yet the specific remedies in the latter answer most of the indications. As long as we are not in possession of a specific remedy for tuberculosis, it will have to be treated symptomatically.

Under the circumstances, to be effective, treatment must be applied in accordance with the clinical manifestations encountered and to a certain extent with the clinical form of the disease. We have seen that each form pursues a course more or less different from all other forms. It would therefore be wrong to treat a patient with abortive tuberculosis in the same manner, and for the same length of time as one with chronic progressive phthisis. Fibroid phthisis demands different treatment than chronic caseous phthisis; febrile cases cannot be treated like those which run an afebrile course. The various complications of the disease, like intestinal, laryngeal, and renal tuberculosis demand special care which the general indications do not satisfy. Preëxisting disease, like syphilis, diabetes, cardiovascular, and renal derangements, etc., alter the course of treatment appreciably. There are also differences in our methods of treatment when we care for a tuberculous child as compared with those applied in adults; but in senile phthisis the indications are not the same as those in adolescents. The indications are even different in cases of young, single women, as compared with married or pregnant women, and during the menopause tuberculosis often demands special treatment.

It is thus obvious that a method of treatment which will suit all cases cannot be formulated. What may be efficacious in one may not be feasible in another, or even harmful in a third. The treatment of phthisis must be individualized to suit the case, it must be elastic and adaptable to the polymorphous nature of the disease and to the various accidents and complications occurring during its course.

**Criteria of Efficacy of Treatment.**—In judging the value of any method of treatment, we must bear in mind some points which are usually neglected while speaking of this subject. The fact must not escape us that the vast majority of cases of tuberculosis manifest a strong tendency to recover under any method of treatment, or even spontaneously. Impressed by the malignancy of the disease in many cases, we are apt to forget the large number of spontaneous recoveries, and when we meet with good results, we are apt to attribute them to the method of treatment pursued, forgetting that a large proportion of patients would have recovered without the treatment.

Discussing the clinical features of abortive tuberculosis, we have shown that this form of phthisis is very common and may not be recognized. When reading about a large proportion of recoveries in a sanatorium which admits only "incipient" cases, or of a drug which is alleged to cure at this stage a certain proportion of cases, etc., we must recall that among these "early" cases, there is a large number with a strong tendency to recovery under all circumstances. To be of real value, a method of treatment must be effective in producing more recoveries than would be ordinarily anticipated.

Even in the forms of chronic phthisis which usually last for many months or years before terminating in recovery or death, the course is not always progressive, continuously advancing. This is evident from the large number of patients who give a history of hemoptysis, cough, fever, emaciation, etc., five, ten, or more years before the onset of the present illness, which was diagnosticated at the time as tuberculosis, but the patient did well. For long years he was able to attend to his work, only being laid up now and then for a few days with an attack of "bronchitis," "grippe," etc., but this last attack has proved persistent. Now, if in this case a proper diagnosis had been made during any of the previous attacks, the prompt recovery would have been credited to the special treatment applied. In fact, many patients tell us that a certain prescription was very effective for years in relieving them promptly, but this time it has failed.

All properly investigated statistical examinations have shown conclusively that five years after the onset of active phthisis about 50 per cent. of the patients are in good or fair physical condition and even able to make themselves useful at their respective occupations, irrespective of what method of treatment was applied. The statistics of results obtained in sanatoriums published by Lawrason Brown,<sup>1</sup> Herbert Maxon King,<sup>2</sup> and others show that patients discharged in the advanced stages of the disease are often found alive and active, five, ten, or even fifteen years later. A physician who keeps careful records and publishes a series of cases in which such results are shown can impress the profession that his method of treatment has done wonders. Yet, it is just what should be expected under any method.

A study of the literature on phthisiotherapy shows that nearly all authors, urging their methods, report certain and almost the same percentages of patients "cured," "disease arrested," "improved," "unimproved," and last, but always least, "dead." Practically all sanatoriums, whether located on high or low altitudes, at the sea-coast or inland, in cold, warm or moderate climates; irrespective of the special method of treatment pursued—indoors, outdoors, or in tents; no matter what the fad or hobby of the attending physician, be it dietetic, medicinal, or specific; they all give the same results if we should judge them by the percentages of reported cures, improvements and deaths as published in their annual report.

During the first year or two after the introduction of new drugs or specifics, physicians report excellent results, as is seen from the literature on creosote and arsenic and their derivatives, ichthyol, cinnamic acid, iodine, tannin, succinimide of mercury, etc. They all cured a certain percentage, arrested the disease in a larger percentage and failed only in very acute or progressive or far-advanced cases. Phthisiotherapy has thus been encumbered with an enormous number of medicaments, which have been lauded by many competent and con-

<sup>1</sup> American Medicine, 1904, viii, 879; Ztschr. f. Tuberkulose, 1908, xii, 206.

<sup>2</sup> National Assn. Study and Prev. Tuberc., 1912, viii, 82.

scientious physicians at one time or another, and condemned with equal vigor by others. According to Rénon the popularity of each drug or method of treatment hardly exceeds three years.

These are in fact the reasons why so many new methods of treatment, drugs, specifics, climates, diets, etc., are annually announced as curative agents for tuberculosis. They all depend on the normal proportion of recoveries which occur under any method. That charming French writer, Louis Rénon,<sup>1</sup> says in this connection: "All new therapeutic methods of treatment of tuberculosis, as long as they are harmless, always give the same satisfactory results. This is an axiom which I should like to have printed with heavy type in all the new books on phthisiotherapy. It is an axiom which may be clinically translated into this simple statement: Hurry and take the treatment as long as it cures; if you wait you may be too late."

The reasons for these therapeutic illusions are found in the above stated facts. The disease is acutely progressive in comparatively few cases. In these, all agree that their remedies are of no avail, and they are not counted in the reported cases. In a large proportion there is a strong tendency to spontaneous cure, and they furnish the recoveries for the special climates, specific and empiric therapeutic agents, for the "milk cures," the "song cure," the "grape cure," etc. In the majority of cases of active phthisis the disease runs an undulating course, with more or less frequent exacerbations of acute or subacute symptoms, followed by remissions in the activity of the process. In some the acute exacerbations are very infrequent, long remissions are obtained, the patient feeling comparatively well for several months and the credit is given to the method of treatment.

**Psychic Influences.**—Persons under the influence of mild alcoholic intoxication are very susceptible to suggestion, and the consumptive who is under the influence of tuberculous toxemia is very vulnerable to auto- and heterosuggestion, as was shown in Chapter XIII. Any new drug, especially when boosted in the newspapers, is apt to relieve him in a remarkable manner. We often meet with consumptives who keep on sinking while under the care of a physician, but for some reason are impelled to change their medical adviser and, though the latter makes no changes in the treatment, the patient begins to gain in health and general well-being. This is usually the result of a new, careful, and minute physical examination by some pedantic physician who subjects his patient to all the diagnostic procedures—inspection, palpation, percussion and auscultation; "gives him the benefit of the latest of diagnostic aids," the  $x$ -rays, the cutaneous or subcutaneous tuberculin test, examines the sputum and urine in the presence of the patient, etc., and usually gives the same directions as those of the former physician, but more minutely; orders the patient to report frequently to see whether any changes are necessary. This is often

<sup>1</sup> *Le traitement pratique de la tuberculose pulmonaire*, Paris, 1908, p. 30.

the beginning of a most remarkable improvement in a case that has been going from bad to worse: The appetite returns, the cough ceases, the night sweats disappear, etc., and they gain in weight and strength.

**Suggestion by Tuberculin Treatment.**—There are many phthisiotherapists competent to give authoritative opinion who are convinced that tuberculin, as generally administered in minute doses, acts more by suggestion than by specific action on the tuberculous process in the lung. We shall revert to this subject while speaking of specific treatment. But meanwhile we want to point out the powers of suggestion in specific treatment as shown in a drastic manner by Albert Mathieu and Dobrovici,<sup>1</sup> who announced to the tuberculous patients at the Andral Hospital in Paris, that a new discovery had been made, a new serum had arrived for the cure of tuberculosis, and that shortly a sufficient quantity of the remedy would be available for those in need of it. The patients had to wait for some time, and when the serum arrived they all rejoiced. The new remedy consisted simply of physiological salt solution, but was given the pompous name *Antiphymose*. Certain patients were told that they were fit subjects for antiphymose, while others were denied the treatment on the plea that it would not do them any good. The selected patients were placed under careful observation and their histories were again recorded minutely, so that all felt that they had been seriously given the first opportunity to benefit by a great discovery. No change was made in the surroundings of the patient and the diet, but all other medication was discontinued.

The patients were greatly impressed by the new remedy and the favorable results exceeded all expectations. Within a couple of days there was noted an improvement in the appetite; those who had fever before showed a normal temperature, and the cough, expectoration and night sweats were ameliorated; those who had hemorrhages ceased bleeding, and even the physical findings in the chest showed distinct signs of amelioration of the process. The gain in weight was remarkable, ranging from 1500 gms. to 2 and 3 kilos. As soon as the injections were discontinued all the old symptoms reappeared.

From personal experience<sup>2</sup> with the culture of turtle bacilli injected by Dr. F. F. Friedmann into patients under my care at the Montefiore Home in New York City, I can say that its effects were practically the same as those of Mathieu's antiphymose. The heightened susceptibility to suggestion of the average consumptive were here vividly illustrated. No one will deny that the vast majority of people, healthy and sick, are amenable to suggestion in various ways, but it must be acknowledged that a group of patients suffering from acute or subacute gout or rheumatism, heart disease in a state of decompensation, of nephritis complicated by dyspnea and dropsy, of ulcer of the stomach, of cancer, or of any other organic pathological entity,

<sup>1</sup> Bull. Gén. de therapeut., 1908, cli, 882.

<sup>2</sup> Fishberg, Interstate Med. Jour., 1914, xxi, 349.

would not be influenced to the same extent by suggestion as were the consumptives just mentioned.

It appears that consumptives in all stages of the disease are susceptible to psychotherapy. I have repeatedly observed marked improvement in the subjective symptoms of patients who were told by their physicians that nothing could be done for them because they are doomed, while the new physician, who was promptly called because of the extreme prostration of the patients, assured the unfortunate sufferers that there was no danger at all, and that only careful treatment was necessary to rehabilitate the lost health and strength, and afterward a short visit to the country would enhance the chances for ultimate recovery. I have seen improvement in a patient after three punctures were made in her chest with a view of inducing an artificial pneumothorax, but no nitrogen was introduced into the pleura because of adhesions. Yet the temperature which had been quite above normal for weeks promptly dropped to normal and the patient felt well. That tuberculous patients, as a rule, improve during the first few weeks or months in a new resort or institution is a well-known fact; and that it is usually not the superior climatic conditions or the different method of treatment that was efficacious in this respect is proved by their relapse into their former condition, or by the aggravation of their disease after the novelty of the new surroundings begins to wear off. This is the main reason why climates "wear out."

**Psychotherapy in Tuberculosis.**—This heightened susceptibility of the tuberculous patients to suggestion is of immense value and assistance to the physician who is the fortunate possessor of a personality which stands him in good stead when handling difficult and intractable cases. But it is a double-edged sword. It also interferes in a large measure with the proper appreciation of the value of any therapeutic procedure, because the patients are apt to be impressed with any new remedy, especially if it has been puffed up by an enthusiastic physician, and promptly improve. But the improvement is only short-lived and within a short time all the old symptoms return, as we have shown.

This psychic trait of the tuberculous is, however, of immense value in assisting all physicians in their efforts to alleviate the more painful features of the disease, provided they know how to take advantage of it. Indeed, the success of many physicians in handling tuberculous patients depends on this point, and it is a fact that therapeutic nihilists fail, as a rule, to give relief to this class of patients. The detailed, often written instructions given by physicians to their patients in sanatoriums, the minute doses of tuberculin administered, the vigilant anticipation of reactions, and the careful inquiry as to the effect on the constitutional symptoms, have all the elements of suggestive therapeutics. Without these details, the institutional treatment of tuberculosis, especially in private and costly sanatoriums, would be a failure.



For these reasons the medicinal treatment of tuberculosis has a place in the therapeutics of tuberculosis. The *materia medica* is of assistance not only in alleviating certain annoying symptoms, as we will show later on, but rational medication also imbues the patient with the idea that something is being done for him during his long and trying disease. Medicinal preparations are also palliative, to be sure, but they often carry the patient over an acute crisis with more or less comfort which could not be obtained otherwise, and they stimulate a hopeful outlook for an ultimate recovery.

**The Indications for Treatment.**—In the absence of specific remedies the therapeutic aims are to increase the natural forces of resistance of the tissues by constitutional treatment and by direct local treatment of the affected lung. The first indication is met by certain general therapeutic measures, the second by the induction of an artificial pneumothorax. In this book the treatment of phthisis is discussed with a view of methodically presenting the subject in the following order:

1. General management of the case.
2. Dietetic management of the case.
3. Institutional treatment.
4. Climatic treatment.
5. Medicinal treatment.
6. Specific treatment.
7. Symptomatic treatment.
8. Local treatment.
9. Treatment of the various forms of tuberculosis.
10. Treatment of the complications.

## CHAPTER XXIX.

### PROPHYLAXIS.

THE recent discoveries in the field of phthysiogenesis have shown that the prophylaxis of tuberculosis is much more complex than the simple formulæ or programs of antituberculosis societies would indicate. A considerable part of the sure preventatives given in popular and technical literature have been shown to be inefficacious or superfluous by the newer teachings of the bacteriology, demography and the clinical phenomena of this disease.

Modern prophylactic measures should differ in accordance with what we aim at attaining. If our aim is to prevent *infection* with tubercle bacilli, we must take different measures than those which are indicated when we aim at preventing *phthisis*, the disease caused by these microorganisms. In our attempts at preventing tuberculosis in children we must resort to different prophylactic methods than when we aim at preventing tuberculous disease in adults. In fact, measures which are likely to prove effective in infants are not indicated in older children, while in adults most of the measures which have been found effective in early life are futile, extravagant and even harmful.

**Prevention of Infection.**—We have seen that the child is born free from tuberculosis, even if its parents are tuberculous at the time of conception or birth. We have also seen that during the first year of life some become infected and that the proportion showing signs of harboring tubercle bacilli in their bodies keeps on gradually increasing with advancing years so that at ten years the vast majority are infected and that at the age of fourteen over 90 per cent. react to tuberculin—an unmistakable sign of having been infected with tubercle bacilli.

We have also shown that during the first year of life infection, if it does occur, is likely to result in an acute or subacute disease which proves fatal in nearly all cases. On the other hand, after passing the age of infancy infection becomes less dangerous, only rarely causing death, though it is liable, when localizing itself in glands, bones and joints, to cause prolonged sickness and end in disfigurement, if the patient survives.

Our main aim is therefore clear. The infant under two years of age must be protected against tuberculous infection at all costs. In families in which there is no tuberculous member this is a simple matter. Impressing the parents that infants acquire tuberculosis very readily, as easily as measles, scarlet fever, influenza, etc., and that a single exposure is liable to result in infection, they can, with reasonable and ordinary care, shelter their young offspring against

the tubercle bacilli. Especially is this an easy matter with mothers who suckle their babies, and do not give them any cow's milk, so that bovine infection is entirely excluded.

An infant is naturally not apt to come in contact with strangers unless those who care for it bring it in their proximity. Realizing that there are so many persons with open tuberculosis who are considered quite healthy, or who consider themselves healthy, it is obvious that in order to positively avoid infection at that age, infants must not be brought in contact with any one excepting the immediate family who are known to be free from the disease.

But it must be remembered that the immediate family includes the grandparents, and they are often suffering from latent tuberculosis. The impression is gaining ground of late that a large proportion of the chronic bronchitis, pulmonary emphysema, asthma, etc., in aged persons, is of a tuberculous character, as was already shown in the chapter on phthisis in the aged. The writer in attempting to trace the source of infection has often found that it was the coughing or expectorating grandfather or grandmother who was responsible for the disease in an infant.

Great care is to be exercised in selecting domestic servants for homes with infants. Especial care is to be taken with the nurse for an infant. She should be carefully examined by a physician, and reexamined if she acquires a "cold" that lasts more than a week.

These simple measures suffice in homes in which there are no tuberculous inhabitants. No infant should be allowed to remain in a home in which a phthisical person resides. Even if the patient is one of the most scrupulous and takes excellent care of his sputum, he should not live in the same home in which an infant is raised. This is a point which, in our efforts to prevent the dissemination of the disease, is often overlooked. Following up phthisical patients, the authorities usually state that a careful consumptive is harmless, as long as he takes care of his expectoration, and permit tuberculous persons to live in the same home with infants. But as a matter of fact the harmlessness of consumptives extends only to adults, and not because they are taking extreme care of their expectoration, but for other reasons which will be given later on in this chapter. As regards infants, no care, however conscientiously exercised, can surely prevent infection. And infection in infants is likely to prove deadly.

The indications are therefore clear. Either the phthisical person or the infant is to be removed. No compromise can be allowed in such cases.

No tuberculous mother is to be allowed to rear her young children, especially during infancy. It has been found that very few infants survive when suckled by a mother suffering from phthisis. The extensive statistics of Weinberg,<sup>1</sup> embracing 5000 families with 18,000

<sup>1</sup> Die Kinder der Tuberkulösen, Leipsic, 1913.

children, have shown that the nearer the birth of the children to the time of death of their tuberculous parents, the higher the mortality among them. Three-fourths of the children born during the last year of life of the tuberculous mothers, succumb; and 90 per cent. of the children born during the last month of life of tuberculous mothers die. The investigations of the present writer<sup>1</sup> among children of tuberculous parentage in New York City have shown practically the same condition to prevail. In addition to the excessive mortality in general, 16 per cent. of the deaths among children under six years of age were due to tuberculous meningitis, as against only 1.27 per cent. among the general population of New York City.

The prophylactic value of separation of the infant from its tuberculous parents is well exemplified by experiences with tuberculous animals. Harlow Brooks<sup>2</sup> shows that among cattle the question of whether or not the offspring becomes tuberculous depends entirely upon exposure after birth. It has been conclusively shown that the calves are very rarely, if ever, infected before birth, but that the slightest carelessness in exposure of the newborn calves to infections leads to certain disaster. It has been found that tuberculous animals may be utilized for breeding purposes and that they may be crossed and inbred with entire disregard of the factor of tuberculosis and purely for the purpose of improving or maintaining the type, provided the calves are separated from the parents immediately after birth.

Similar measures have to be taken in cases of newborn infants of tuberculous parentage. If the mother is tuberculous the infant is to be removed immediately after delivery, and should not be allowed in her proximity during the first two years of life. If the father is phthisical, he should be removed from the home, as long as there are infants under two years of age. In some cases the alternative of removing the infant may be more feasible. Bernheim induced three tuberculous mothers who had twins to separate with one child each, while retaining the others in their homes, though healthy wet-nurses were employed to suckle the babies. The three isolated children remained healthy, while the three which were raised at home succumbed to tuberculosis.

It is noteworthy that improvement in the sanitary and hygienic conditions, which are so effective in preventing phthisis in the adult, as will be shown later on, are not of any value in the case of infants. As has been pointed out by Römer, it was found that scrupulous attention to hygiene and sanitation of the stable, such as proper construction, ventilation, cleanliness, etc., hardly has any influence on the prevalence of tuberculosis in cattle and that only strict isolation of the sick from the healthy animals is effective. Primary infection in infants appears to follow the same law: Exposure of an infant, even in an ideal home, may result in fatal tuberculosis, while life under

<sup>1</sup> Archives of Pediatrics, 1914, xxxi, 96, 197.

<sup>2</sup> Amer. Jour. Med. Sci., 1914, cxlviii, 718.

adverse conditions will not produce tuberculous disease, unless there is a source of infection, which is usually the human consumptive and rarely the milk derived from tuberculous cows. In the development of phthisis in adults hygienic and sanitary conditions play, however, a very important role.

The prevention of bovine tuberculosis is not to be neglected. When an infant must be hand fed, the milk should be carefully selected. In large cities the only drawback is the cost. Certified milk is everywhere available, but it is rather expensive and prohibitive for the vast majority of the population. For this reason all milk that is not derived from a source known to be safe, is to be pasteurized or better yet, sterilized.

These simple measures are to be taken with a view of successfully preventing primary infection of infants under three years of age. They can be easily carried out by any family which has some degree of economic independence. In families which are to some extent hampered because of economic stress, the State is to interfere. Health Boards, which are busy protecting adults against *infection* to which they are hardly susceptible, could perform really useful service if they concentrated more and more along these lines. The mortality during the tender age of infancy, which has hardly been influenced by the campaign against tuberculosis, would be reduced to a minimum. Moreover, massive infection, which is apparently responsible for phthisis in the adults who have survived it during infancy, may thus be largely prevented.

**Prophylaxis in Children over Three Years of Age.**—When the child begins to walk around and comes in contact with many people, prevention of infection is not simple. The parents, especially those who cannot afford a maid for each child—and they constitute the bulk of population—lose control over their children, unless they are prepared to keep them altogether from contact with strangers and this is not feasible for obvious reasons. Later when they go to school, they are bound to come in contact with other children and adults and it is altogether impossible to prevent their meeting tuberculous individuals, no matter what the economic condition of the parents. It is thus clear that it is altogether impossible to prevent tuberculous infection among children over four or five years of age.

But, as was shown in Chapter XXIV, infection in children over four years of age is usually relatively harmless. Either no disease at all occurs, or rarely, tracheobronchial adenopathy results, which is serious only on exceedingly rare occasions.

Available evidence tends to show that in infants infection is usually accomplished within the family—tuberculosis is exceedingly rare in infants who live in homes in which there is no phthisical member. When this is the case, we may trace the infection to someone living in the house as a lodger, or to some relative or friend who visits the home and comes in intimate contact with the infant, thus causing

massive infection. With children of play and school age, the opportunities for intimate contact with adult strangers are scarce; they are not taken in the arms, not kissed indiscriminately, etc., and even when infection takes place it is from another child, a playmate, etc., is slight, and not as massive as it is apt to be in infants, who are infected from adults.

There is abundant clinical evidence of the relative harmlessness of infection of children over four years of age. One has but to consult the mortality returns in any country to convince himself that between three and fifteen years of age the mortality rates from tuberculosis are comparatively low, despite the fact that over 90 per cent. of the tuberculous infection of humanity takes place during this period of life. Comparing the results of infection during the first two years of life, and those taking place between four and fifteen years of age, the contrast is striking and convincing (see p. 359). Neither acute tuberculosis nor chronic phthisis of the adult is common in children of school age. Thus, among 925,000 children examined by the medical school inspectors in New York City during the school year September, 1914 to June, 1915, only 68 were found tuberculous.<sup>1</sup> When we bear in mind that each was examined by physicians and nurses once in six weeks on the average, and that a complete physical examination was made of all children three times during the course of the elementary school year, and that a cough noted by the teacher was sufficient to refer the child for examination, it is obvious that not many suffering from tuberculosis were overlooked.

Under the circumstances, we may conclude that no matter what the cause is, infection of children under school age is comparatively harmless, and that inasmuch as experience has taught that everybody is bound to be infected with tubercle bacilli, the best that can happen is that infection should occur at the age period of four to fourteen years. The primary mild infection at that age, as we have shown above, practically vaccinates humanity against more severe infections in later years. Otherwise, all adults would be as susceptible to tuberculosis as are guinea-pigs, or the indigenous races of Central Africa.

Our efforts are therefore to be directed, next to the prevention of contact of infants with tuberculous persons, at the prevention of massive infection of children. This can be done within certain limits by preventing children from associating with individuals suffering from open tuberculosis. The danger lurks mostly in adults, because children expectorating tubercle bacilli are exceedingly rare.

**Prevention of Reinfection.**—It thus appears that the bacilli infecting children remain dormant within the body, and cause no disease, as long as there are no predisposing or exciting causes. We know that under certain circumstances these dormant bacilli activate and cause disease by metastatic auto-infection. This is mainly seen in cases in

<sup>1</sup> Weekly Bulletin of the Department of Health, City of New York, 1915, iv, 289.

which, owing to defective nutrition, or some intercurrent disease, notably measles, whooping cough, typhoid, etc., the resistance is reduced, and an exacerbation of the tuberculous process takes place. Moreover, it appears that the younger the child, the more is the anergy thus induced likely to be followed by active tuberculous disease. The indications are therefore clear—young children and infants are to be sheltered against the endemic diseases. Special care is to be exercised in this direction with children of tuberculous parentage, who have in all probabilities suffered from massive infection. This class of infants is to be scrupulously shielded against measles, whooping-cough, scarlet fever, diphtheria, etc. If these diseases are bound to attack them, it is best that it should occur after they have passed the fourth year of life.

During convalescence after one of these endemic diseases, the child is to be given special care, with a view of preventing metastatic auto-infection while the body is in a state of anergy; in other words, susceptible. This may be done by either taking the child to the country, preferably to the seashore, for a few weeks or months, till it has completely recuperated; or when it must be kept at home it should be given proper nourishment, and kept outdoors the greater part of the day, and it should sleep in a room with open windows.

**Prophylaxis in Adults.**—Prophylaxis in adults is no more a problem of infection. It may be taken for granted that everyone who has passed through the first fifteen years of life, especially in a city, has been infected with tubercle bacilli. The fact that he shows no symptoms and signs of disease is no proof that he has escaped infection, as was already shown. In adults, the problem is the prevention of disease, of phthisis. I believe that a considerable portion of the inefficacy of the campaign against tuberculosis is due to the lack of appreciation of this distinction between infection and disease.

This fact is based on the newer investigations in phthisiogenesis, which have conclusively proved two points:

1. That chronic phthisis in the adult, of the type that creates most of the tuberculosis problem, never occurs immediately after a primary infection; if disease occurs at all soon after a primary infection, it is of the acute types of tuberculosis of the lungs or of other organs. Indeed when disease follows immediately after a primary infection of an adult it is almost invariably deadly, as is seen in tuberculosis of primitive peoples who have not been exposed to infection during childhood.

2. Infection with tubercle bacilli, whether it causes disease or not, renders the body immune against further and renewed exogenic infection with the same virus. Inasmuch as nearly all adults have been infected with tubercle bacilli during their childhood, they are immune against reinfection with bacilli which may be eliminated by tuberculous persons. The phthisical manifestations in adults are attributed to the infection during childhood, just as the tertiary mani-

festations of syphilis are late results of the original infection years ago, though the body is immune against renewed exogenic infection with the same virus.

If this was not a fact, practically all the workers in hospitals for consumptives would succumb to the disease: all consorts of tuberculous persons would acquire the disease. One has to consider that of women married to, and living with, husbands suffering from active syphilis, hardly any escape infection. But we see thousands of tuberculous persons living with consorts, having children with them, yet the unaffected consorts remain in good health, as we have already shown in detail (see p. 110).

It is therefore a vain effort to follow up tuberculous persons, push them from pillar to post, interfere with their employment, as has been done in many cases with a view of preventing infection of fellow-workmen. If these individuals cannot infect their husbands or wives, as the case may be, despite the intimate contact, they are surely not a menace to their fellow-workmen.

This fact is now beginning to be recognized by those who are well-informed about the recent progress in our knowledge of phthisiogenesis. There has been manifesting itself a reaction against the absurd and cruel phthisiophobia which has been rampant for about twenty-five years. Baldwin<sup>1</sup> says: "Adults are very little endangered by close contact with open tuberculosis, and not at all in ordinary association. . . . It is time for a reaction against the extreme ideas of infection now prevailing. There has been too much read into the popular literature by health boards and lectures that has no sound basis in facts and it needs to be dropped out and revised."

**Prevention of Phthisis.**—It appears that in the eager chase after the bacteria, which could never be entirely destroyed, we have forgotten that only a small portion of those infected develop phthisis, while the rest are apparently benefited by the infection. Some recent writers have not hesitated to apply the term *benevolent infection* to those who have been fortunate in acquiring tuberculosis during later childhood and have thus been immunized against primary infection after fifteen years of age, when the disease produced by a primary infection is apt to run an acute and fatal course. Otherwise, we would all succumb to the acute and fatal forms of tuberculosis.

Phthisis is a disease occurring in persons who have been infected with tubercle bacilli many years before the outbreak of the disease. It is due to reinfection. But available evidence appears to point in the direction that the reinfection occurs from within, that it is metastatic; the bacteria which have remained dormant for years are slowly or suddenly reawakened into activity and they produce new lesions; and that exogenic reinfection is exceedingly rare, if at all possible.

We know that certain conditions favor a reduction in the normal

<sup>1</sup> Johns Hopkins Hosp. Bull., 1913, xxiv, 220.



resisting powers of the body and permit the proliferation of the dormant bacilli. Among these, inferior sanitary, hygienic, and economic conditions stand out preëminently. We have seen that the rates of wages, the number of rooms in which a family lives, the character of the work pursued by an individual, etc., have a strong influence in the direction of enhancing or preventing the evolution of phthisis. For this reason, the philanthropic agencies may do more toward the prevention of phthisis by concentrating their attention on improvements along economic lines of reform than by sending agents to tell adults that it is dangerous to remain in the proximity of a consumptive. Labor unions do better by exacting higher wages and shorter hours than driving unfortunate phthysical persons from their places of employment, as is being done of late in New York City.

**Phthisiophobia.**—Phthisis is undoubtedly an exacerbation of dormant tuberculous processes in the lungs; its entire clinical course is undulating, with periods of quiescence interrupted by periods of activity. These acute and subacute exacerbations may be prevented by careful attention to the general health of any individual who shows the least tendency to phthysical disease. Such individuals should not be hounded, refused employment, etc. They are to be helped along in the direction of securing easy work during the quiescent periods, so that they may be self-supporting and self-respecting. The words of an intelligent and observing consumptive on this subject are to be borne in mind by social workers, who of late seem to know more of the etiology and prevention of tuberculosis than those who have made a special study of the subject. Says the American historian, William Garrot Brown, in his *Confessions of a Consumptive*:

“The public depends for protection from such danger as our continued existence involves, not on its own exertions but on ours. To render that protection we must burden ourselves with both expense and trouble. We must incessantly take, for the sake of the public, precautions which are disagreeable and costly; and meanwhile a great part of the public is, by its attitude toward us, steadily tempting us, and even sometimes fairly compelling us, if we would live to discontinue these precautions and go on as if there were nothing the matter with us. The folly and stupidity of this attitude it is impossible to overstate. It is of itself by far the chief cause and source of the persistence of this scourge.

“Known and recognized and decently entreated, we are not dangerous. Shunned and proscribed and forced to concealments we *are* dangerous. Victims ourselves of this same regime of ignorant and self-deceiving inhumanity, we are called on every hour of our lives for a magnanimous consideration of others. Society can hardly find it surprising or a grievance if our human nature should sometimes weaken under the strain of the incessant provocation it endures from this strange working of human nature in general. Why should we alone be expected to be guiltless, always to our own cost and sacrifice, of

that very form of man's inhumanity to man, from which we ourselves are suffering more than anybody else? Yet I can honestly attest that the vast majority of us are guiltless of any merely resentful offense; that, as a rule, when we fail to protect the public it is only because the public compels us to disregard its interest, its safety. This is what I earnestly entreat the public, for its own sake, candidly to consider.

"Candidly means fully. If the public is to be safe from us, if the public is to continue to have our protection from that against which it failed to protect us, then the public must make it possible for us to get—it must certainly cease to make it impossible for the mass of us to get anything except by subterfuge—what we must have to live. We are neither criminals nor mendicants. We do not ask favors, we merely revolt against a mean and stupid oppression. We revolt against ignorance and against a lie. The public would get rid of us, and thereby makes us inescapable. It would pretend, and would have us pretend, that we are nowhere. It thereby insures that we shall be everywhere. It proscribes us and thereby admits us."

If the average consumptive was not shunned by adults; if he was permitted to work unmolested after he is cured or the disease is arrested, or quiescent, allowing him to earn his livelihood, a considerable part of the economic stress caused by this disease would be done away with. If the tuberculous individual is told that he is only a menace to infants, less dangerous to children, and not at all dangerous to adults, he will surely take all precautions against infecting those who may be harmed by it.

But at present the State, municipal, philanthropic and social agencies that send out representatives telling those who live with consumptives that the patients must be shunned, and incidentally conveying the information that a careful patient, *i. e.*, one who takes care of his sputum, is not at all dangerous even to infants. Some patients in New York City are actually dreading lest their names will be reported to the authorities, and they will be pestered by those well-meaning nurses, physicians, social workers, etc. Instead of telling the patient that he is only a menace to infants, and that he must keep away from them, they often visit his place of employment and the result is that the unfortunate patient is soon without a job and starving.

The results of these methods of phthisiophobia are seen in the fact that the number of infants which succumb to tuberculosis has not decreased even in Germany where antituberculosis agencies have been most active; that the number of persons infected with tubercle bacilli has not decreased is clear when we consider that over 90 per cent. of humanity react to tuberculin.

I do not want to be understood as speaking unfavorably of all prophylactic measures against tuberculous infection of adults. There are many especially among the richer classes in cities, and in suburban and rural districts, who have escaped infection during childhood, and they should be protected. It is in fact well known that tuberculosis, when occurring

in these classes is often of an acute type, just as it is in the indigenous races of Central Africa, or the Esquimaux. They should be protected against the sputum indiscriminately expectorated by consumptives, and against droplet infection when coming in contact with persons suffering from active phthisis. But with the city-bred people, especially those who have survived in the congested parts of cities, or the slums, there is hardly any danger that adults will be infected with tubercle bacilli. They have been infected during childhood; vaccinated and immunized against additional infection. But it is just among these that the strong efforts are made to prevent exposure of adults to infection. The irony is that their infants are usually neglected by the social forces working in the antituberculosis campaign.

Just as the cattle breeders have found that the control of tuberculosis is mainly a matter of prevention of infection of newborn calves, and that adult cattle may be disregarded, so must we act with humans. To prevent infection, newborn infants must be protected while children over ten and adults need no special measures, especially those who have been raised in cities.

**Disposal of the Sputum.**—In our attempts at preventing infection, the disposal of the sputum expectorated by phthisical patients is more important than any other prophylactic measure. The saprophytic bacilli are distributed in virulent form only from one animal body to another. Exceptionally, the source of the bacilli is a domestic animal, mainly milk from tuberculous cows, but in the vast majority of cases the source of infection is sputum expectorated by phthisical patients.

For this reason the rigorous laws prohibiting indiscriminate expectoration which enlightened communities have inaugurated are fully justified, and they ought to be more rigorously enforced. It should be made clear that tuberculosis is not the only disease which is transmitted by expectoration, but many other diseases are dangerous in this regard, so that nobody ought to spit on the floor of a house or public place. Furthermore, there are many tubercle bacillus "carriers" who do not suffer from the disease which they are liable to transmit, especially to infants and children. The fact that indiscriminate expectoration is enforced irrespective of the question whether the offender is tuberculous or not, makes it easier to exact it from the phthisical patients, who do not like to be stigmatized.

In the case of children, especially infants, it is not only sputum which is dangerous, but also the droplets flying out of the mouth and nose during the acts of coughing, sneezing, and talking. For this reason a consumptive should not associate with infants, even if he is careful with his expectoration. Droplet infection may prove disastrous to infants. In the case of adults, coughing and sneezing are hardly dangerous. We have already mentioned Saugman's conclusion that it is not dangerous for adults to be coughed at by a tuberculous patient (see p. 110).

**Cuspidors.**—The disposal of the expectoration is therefore an important problem, and it has been suggested that the best means of rendering it harmless is that it should invariably be deposited in some form of cuspidor.

Floor cuspidors in rooms, especially in public places, are a nuisance; they cannot be tolerated in any decent home for both sanitary and esthetic reasons. They are unsightly, and just as much of the sputum is often deposited around the vessel as within it. Flies, cats, and dogs are frequent visitors and with mouths or legs covered with sputum may proceed further in their quest for food, and deposit the bacilli on food which is subsequently used by the inhabitants of the house. The elevated cuspidors, of which we find such beautiful illustrations in a certain variety of books on tuberculosis, may be good for certain institutions, especially those harboring advanced consumptives, but they should not be, and are not, used in homes and public buildings. They are also an invitation to spit; they provoke expectoration in persons who otherwise would not do it. This is the reason why they are hardly seen anywhere, except in institutions.

The pocket sputum flasks are objectionable for other reasons. Their variety is great, if we are to judge by the large number illustrated in popular books on the prevention of tuberculosis. The ingenuity of the designers or inventors is noteworthy and could have been used to better advantage in other directions. They are, however, not used outside of institutions to any noticeable extent. I fancy that a person who would take out a sputum flask, even one of those which look like cigar boxes, lunch boxes, etc., and spit into it within the sight of people in a public place, would create a miniature panic among some who have read popular literature on the prevention of tuberculosis.

They are objectionable for another reason. No matter how wide-necked they are made, the patient must apply his lips to the mouth of the flask if he wants to deposit the sputum within it. The result is that part of the sputum sticks to the lips or mustache and beard, and this must be removed with a handkerchief. Even if all male patients would consent to shave clean it would not help. I have observed that the lips are very often covered with sputum after the patient has expectorated into any of these flasks.

In institutions they should be used, and the ones made of pasteboard, kept in a tin frame-holder, are the best. Patients in the advanced stages of the disease should use them at home in case they expectorate large quantities of sputum.

But I can see no reason for urging them on patients in the incipient stages of the disease, expectorating but little sputum. Physicians trying to imitate legislators who pass laws which they know cannot be enforced, defeat their own ends. We cannot induce a patient to carry a sputum flask with him, no matter how fine and deceptive its construction may be, and to use it in public. I have also known some

patients in the incipient stages of the disease who left sanatoriums because they could not tolerate their fellow-sufferers walking around with sputum cups in their hands. Advanced patients are hardened in this respect as a rule.

Patients in the incipient, or quiescent, stages of the disease can empty their chests in the morning into cuspidors containing some cheap disinfectant. It should soon be emptied into the water-closet. Urging them to burn it is usually a vain effort, if only because there are no facilities in modern homes for the purpose. Those expectorating considerable quantities may efficiently dispose of their sputum by the use of paper napkins, as suggested by Landis.<sup>1</sup> Toilet paper will also answer the purpose. Several thicknesses are folded once, so as to receive the sputum; the paper is again folded and the ends folded over so as to enclose the expectorated material, and then placed in a grocer's bag (about 6 by 12 inches). The bag can be pinned to the side of the bed or clamped to the small bed table. Several times a day, depending on the amount of sputum, the bag and its contents should be burned, if there are facilities for the purpose. The folded paper pockets containing the sputum may, however, be disposed of by dropping them singly into the water-closet and flushing it immediately.

There is no question that there are valid objections to the handkerchief, though it is not as strong a menace as some writers would lead us to believe. But the average patient will use nothing else for reasons already stated. Portable sputum cups are used only in institutions and in homes, but, despite the agitation in their favor, we fail to meet persons in the streets or public places of any large city in the world carrying and expectorating into them, although we know that thousands of consumptives are everywhere. Even if it is a compromise, we must submit to the inevitable and permit patients to use handkerchiefs. It is best that they should be made of gauze or cheap cotton which may be destroyed after use; or they may be of Japan paper which may be deposited into the water-closet which is immediately flushed. If made of better material, the handkerchief should be boiled before washing. Boiling is a better and surer bactericide, especially of tubercle bacilli in sputum, than any chemical disinfectant.

**Duties of the Community in the Prevention of Phthisis.**—In its demands on the consumptive to shape his life in such a manner as to prevent the dissemination of the disease, the community must not neglect its own duties to the unfortunate individual, who is suffering to a great extent because of conditions which the authorities have permitted to prevail. The community must not only provide shelter, proper nourishment and medical attendance for those patients who are not in a position to procure it at their own expense, but must also see to it that the conditions favoring the development of phthisis should be eliminated.

<sup>1</sup> Hare's Modern Treatment, Philadelphia, 1910, i, 740.

Laws regulating the sanitary and hygienic conditions of dwellings for the working people, among whom the proportion of phthisical patients is highest, should be passed and rigorously enforced. Tenement house laws, passed and enforced, have a greater influence on the reduction of the morbidity and mortality from consumption than all the lectures delivered in and out of season to social workers, policemen, teachers, and workmen, on the perils of the tubercle bacilli and the best means of killing them. The demolition of the old-style tenements with numerous rooms without windows, has saved many more persons from developing phthisis than all the sanatoriums which are supposed to isolate the sources of infection, but which, in fact, exclude those in the advanced stages and permit them to come into intimate contact with infants and children. The abolition of the sweat-shops in New York City deserves more credit for the prevention of phthisis than all the leaflets which have been distributed by so many overlapping agencies, each eager to get at the persons who cough as a result of tuberculosis or some other disease and "follow them up."

Light and well-ventilated dwellings and workshops are of prime importance in preventing phthisis, and the community in which there are no rooms without windows and no sweat-shops or factories which are dark and badly ventilated has the least consumptives to care for.

Good wages and short hours, allowing good nourishment and time for outdoor exercises and recreation are important in the control of phthisis.

**Marriage of the Tuberculous.**—The problem of marriage is one which the physician often has to solve for his patients. We frequently have to answer the question whether a non-phthisical consort should continue to live with the phthisical partner; or whether a tuberculous patient, in any stage of the disease, may enter the married state. Answering these questions involves a consideration of several factors. The dangers of transmission of the disease to the non-phthisical consort; the dangers to the potential offspring; and the effect of the married state on the patient.

The dangers of transmission of the disease to the consort are negligible. We have seen that statistics prove that the unaffected consorts of consumptives are no more liable to become phthisical than others of the same age and social condition (see p. 110). The unaffected consort has undoubtedly been infected during childhood, and reinfection is not likely. Whether he or she will develop phthisis depends on factors other than reinfection from the patient. The conclusion is therefore justified that as regards transmission of the disease alone there is no more danger in marriage of phthisical patients than in cases of cancerous or diabetic patients. Our answer is to be about the same as when two persons who had both been previously infected with syphilis ask whether they are permitted to marry.

The danger to the children that may result from the union is enormous. If the newborn child will remain in the proximity of the

phthisical parent, it will most likely become infected during infancy and succumb. Under the circumstances, unless they are satisfied to remove the child immediately after birth and not see it till it has passed the first two years of life, phthisical patients should not procreate. This is a point which cannot be emphasized too strongly to tuberculous patients who are married or contemplate marriage. It is especially dangerous for an actively phthisical woman to raise infants. They will, we can say almost without exception, acquire the disease and succumb during the first year of life.

The effects of the married state on the patient is different in men as compared with women. On the average male patient in the incipient or moderately advanced stages of the disease, sexual intercourse has the same effect as on the average person who is not in perfect health. If he indulges moderately it does him no harm at all; in fact, it may be beneficial because it prevents brooding over enforced abstinence which is often seen among all classes of men. It also precludes venereal complications which may have an effect on the phthisical process.

With women things are different. As long as they do not become pregnant there are no strong and valid reasons against married life. In fact, among the working classes the married consumptive woman is better situated than the single who soon after becoming tuberculous also becomes a dependent; and if she has no family to care for her, is doomed. But pregnancy, childbirth, and lactation are functions which are of grave augury for a consumptive woman. Occasionally we see that during the pregnant state the tuberculous process in the lung improves, and the general condition of the patient is strikingly ameliorated. But in the vast majority of cases, soon after childbirth there is an acute or subacute exacerbation of the disease and the patient succumbs within a few months.

Married tuberculous women are therefore to be given detailed instruction on the proper methods of prevention of conception. If they become pregnant the induction of abortion is indicated and justified both for the sake of the prospective child, which is bound to become tuberculous unless removed from the proximity of the mother immediately after birth, and for the sake of the mother, who is liable to succumb to acute or subacute tuberculosis soon after childbirth.

The demands made by some enthusiastic advocates of eugenics that tuberculous persons should be prohibited by law from marrying, has no scientific basis in view of what has been stated above. The race is not in danger of deterioration because of children derived from tuberculous stock. We have already mentioned that tuberculous cattle have been used for breeding purposes by removing the calves immediately after birth. We see no reason why this should not hold in human beings. Moreover, prohibition of legal marriage does not exclude extramarital sexual intercourse and childbirth with their concomitants. Free instruction on the means of prevention of con-

ception is more likely to eliminate phthisical stock and thus prove of eugenic value, than prohibition of marriage.

A patient presenting himself or herself with the problem of marriage should be explained the situation along the lines just detailed and if he or she is intelligent, we may rest assured that the action will be reasonable for both the married couple and the community. The ignorant and reckless will not consult us in such matters and if they do, they will not follow instructions. For this reason, they should be left out of consideration in discussions of this kind. One thing I always insist on with my patients: The unaffected partner must be informed about the true state of affairs and given the choice. Very often it will be found that a good woman will greatly help along a consumptive toward a recovery which could not have been attained if the patient had remained single; or that a female patient will recover after marriage to a man who gives her a good home, proper food, etc.



## CHAPTER XXX.

### GENERAL MANAGEMENT OF THE CASE.

**Should the Patient be Told that He is Tuberculous?**—The diagnosis of pulmonary tuberculosis having been definitely made, there arises the question whether the patient should be told the true nature of his disease. Many physicians are inclined to keep him in ignorance as to the true state of affairs, and to tell him that he is merely affected with a “mild bronchial catarrh,” “pleurisy,” “a protracted cold,” etc. Very often a patient is brought to the office by relatives and friends who beg the physician that in case tuberculosis is diagnosed, the patient should under no circumstances be told the truth.

There are many valid reasons against such a procedure. From the standpoint of the physician's personal interests, it is bad practice. It is always to be borne in mind that the patient will sooner or later find out the truth and blame his doctor for deception or, more often, accuse him of ignorance and claim, with considerable justice, that had he been informed in time he might have taken better care of himself.

But there are reasons of more importance than the doctor's interests for telling the truth to every patient on such occasion. It must never be lost sight of that tuberculosis is transmissible, particularly to infants and children, and that the patient must be warned against the possibility of disseminating the seeds of the disease. This can only be done by telling the patient the true state of affairs and giving him details of the principles of prevention. Moreover, the average patient knows that, in many cases, the chances of recovery diminish with the advance of the disease, and negligence in informing him of his opportunities at the earliest possible time may prove disastrous. We do not know of any quick cures, and the coöperation of the patient is absolutely essential. He can only take proper care of himself and those around him when he knows the true situation.

It is noteworthy that relatives and friends who have requested a physician to keep the patient in ignorance of the fact that he is tuberculous are always grateful in the end when he is tactfully informed of the truth.

Irrespective of requests of friends and relatives the patient is to be told plainly and unequivocally that he suffers from tuberculosis. In really incipient cases this can be done in several instalments because it usually requires several examinations to make a positive diagnosis. But when finally told, it is to be emphasized that he is in the incipient and curable stage, and assurances given that in his case the prognosis

is very favorable. But it must be insisted upon that the patient's cooperation is absolutely essential to attain a cure. An intelligent patient may be given details of the nature of the disease and it may be pointed out that his own determination to follow instructions is of more importance than all the medicines and climates; in fact without his own cooperation, he is lost even if he consults the best known specialists, enters the most famous sanatorium, or emigrates to any climatic resort. It is a striking fact that nervous and excitable patients who are expected by their relatives to break down on hearing the truth, resign themselves to their fate and often display a courage and determination worthy of heroes.

"Unless we carry conviction to our patients," says Arthur Latham,<sup>1</sup> "they are unlikely to put up with the restrictions which are inevitable to proper treatment. It is a disastrous thing to talk about a "weak spot" in the lung. It is our duty, in an overwhelming proportion of cases, to state his position frankly to the patient, to explain intelligibly the reasons for the treatment prescribed, and the possible penalties which may have to be faced if our advice is neglected. If we can convince our patient, we shall in all probability have won his loyal cooperation, which is half the battle: if we fail to convince him or get him to see the reasonableness of our advice, we cannot expect to find treatment carried out with sufficient earnestness and consistence to be of real value."

The suggestion has been made by Penzoldt<sup>2</sup> that the dose of truth given to the patient should be in inverse ratio to the seriousness of the case—the less the chances of recovery, the smaller the dose of truth. In incipient and hopeful cases the whole truth is best, but the term "consumption" should be avoided in all cases; "tuberculosis" is a term which covers everything for the patient, though as we have seen, it is not exactly correct scientifically or clinically. But in the popular mind it has been of late considered a hopeful and curable disease, if taken in time. Some patients may be told that when neglected "tuberculosis" may turn into consumption.

As Abraham Jacobi<sup>3</sup> well says: "When a patient strikes a doctor who recognizes a human being in the forlorn creature before him he is told that he has tuberculosis. When he addresses a young colleague, an immature colleague, a colleague satisfied with and gratified by the possession of a diploma and who likes to exhibit his knowledge and authority, he is told he has "consumption." "You have tuberculosis. If it were to get worse it would run into consumption. But cases of tuberculosis may and often do get well, so there is no reason for despair."

It is different with advanced and hopeless cases. They present themselves asking whether their cough is really due to consumption

<sup>1</sup> Practitioner, 1913, xc, 38.

<sup>2</sup> Handbuch der Therapie, 1910, iii, 205.

<sup>3</sup> American Medicine, 1905, x, 1063.

and it is at times a pity to tell the unfortunate patients the true state of affairs; not unless we are not averse to shortening their days. Still, for obvious reasons it is always imperative that some relative or friend should be told the truth. Similarly, in cases of acute or subacute pulmonary tuberculosis, or in progressive cases with complications, such as those suffering from diabetes, tuberculosis of the kidneys, etc., in addition to the active pulmonary lesion, it is often advisable to console the unfortunate and doomed patient if he likes it, by telling him that the prognosis is excellent.

**Economic and Social Conditions.**—In outlining the treatment to be pursued, the social and economic condition of the patient is always to be borne in mind. It is not advisable to tell a patient of limited means that a certain private sanatorium, or a climatic resort in a distant part of the country, is good for him. He is likely to brood over the fact that owing to his poverty he is lost, when in fact he could get along very well at home or in the neighborhood of his city. Well-to-do patients may be sent out of town with only suspicious symptoms and signs of the disease on the principle of some physicians to treat all "suspects" as tuberculous until proved to be free of the disease. The rest during the vacation does them good; in fact, they usually need it. But the patients with limited means should never be treated in this manner. In them only a positive diagnosis of tuberculosis should be the criterion for radical treatment.

**Relation of Physician to Patient.**—A great deal has been written about the relation of the physician and his tuberculous patient and it has been repeatedly stated that the former must possess certain qualifications which, if taken seriously, would exclude 99 per cent. of practitioners from the category of physicians competent to handle an ordinary case. According to one writer, the physician must possess no less than an extraordinarily strong personality, immense will-power to impress it on his patients, unusual teaching ability, fervent enthusiasm and unremitting interest, etc., if he is to meet with success.

Evidently these requirements are such as all ideal physicians should possess if they are to be fit for successful practice. The truth is that in most cases it is quite easy to gain the confidence and coöperation of the patient, if this is at all obtainable. The main problem is to retain it for the long period of time it takes until the termination of the case. This is especially true of chronic phthisis which runs an undulating course with accidents (hemorrhages, fever, anorexia, etc.) which come and go unexpectedly and are liable to shatter the most implicit confidence. This is one of the reasons why tuberculous patients, next to those suffering from venereal diseases, are the best prey for quacks and charlatans.

My observations lead me to the conviction that the average tuberculous patient can be easily managed and his confidence retained for an indefinite time when we appeal to his reason. It is a grave mistake of many superintendents of public sanatoriums who try to obtain the

coöperation of their patients by keeping them in constant fear of punishment—expulsion. As one patient told me, the superintendent inflicted severe punishment on patients for small infractions of the rules of the institution because for these dependent patients the only hope of recovery was the sanatorium. Such severity does not at all help along in gaining the confidence of patients. I know of public sanatoriums in which the patients are always coerced into obedience of the rules and to submitting to prescribed treatment, but they do not discharge the proper proportion of cured patients and a very large number leave the institutions of their own volition before the physicians discharge them.

To a certain extent the patient treated by his physician at home is more amenable to reason than those in public sanatoriums. The physician in private practice is in a position to individualize his cases and more easily persuade them that their only chances for recovery lay in their implicit obedience of orders. When the patient is told the reason why we want him to rest the greater part of the day for weeks or months; why we want him to eat certain kinds and quantities of food; why we want him to submit to the operation for artificial pneumothorax, etc., he is more likely to submit than when we threaten him. All this can be done with alleged ignorant patients, who usually have more common sense than they are credited with, as well as with the intelligent and cultured. In fact, the former are, as a rule, more tractable than the latter. We must always remember that these patients make great sacrifices for months, and need consolation and encouragement which only the reasonable physician is able to bestow.

**Personal Hygiene.**—The first instructions given to the patient are as regards his personal hygiene. This can best be done only after careful inquiry into his daily habits which, as a rule, are found not to have been exemplary; otherwise he would not have been likely to develop phthisis. To be successful, it is necessary to enter into the smallest details of every-day life and most patients appreciate it greatly.

Treating patients in cities, after deciding against a sanatorium, it is of immense importance to ascertain their home surroundings. A call should be made on the house of the patient to see whether it is fit for a tuberculous individual, and especial attention should be paid to the location of the sleeping room, its size, windows, exposure, etc. In case these are not found satisfactory, moving should be urged, preferably to the outskirts of the city or a suburb. Details are given in Chapter XXXII.

In our attempts at adapting the patient's mode of life to the therapeutic indications, we meet with great obstacles when trying to impress him with the urgency of cessation of work, physical and mental, and it is particularly difficult to persuade patients with mild lesions showing few constitutional symptoms. They are convinced that work does them no harm. The poor point to the necessity for providing

for themselves and those dependent on them, while the well-to-do are apt to be even more intractable in this regard. They must not neglect their business, they must finish some task they have undertaken, they are deeply absorbed in some studies; they must continue at college until graduation, etc. But the careful physician is not moved by these pleas and points out to the patient that just because he is in such good physical condition the prognosis is so good. But should he continue working physically or mentally, the disease will surely make inroads on his vitality and the chances of ultimate and complete recovery will vanish. Whether he "leaves" the city or not, the patient may be induced to take a complete vacation with all the separation from the activities of life a vacation entails, but without any of its pleasures. The details about rest and exercise are given in Chapter XXXI.

**Baths.**—The mortal fear for "colds" entertained by many is accentuated as soon as the diagnosis of tuberculosis is made and one of the first results is that the patient ceases to bathe. In many advanced cases, or even in incipients who suffer from profuse nightsweats, large patches of pityriasis versicolor are to be seen on the skin of the neck and trunk. When told that bathing will remove it, women are easily induced to take frequent baths. But all are to be explained that bathing improves the circulation, activates the skin and invigorates the individual. It must be insisted upon that the patient bathes frequently and follows it up by vigorous rubbing of the skin with a rough towel.

The question of cold baths in tuberculosis has been very much debated. In some institutions, cold baths and frictions are the chief elements of the cure. They are urged for the purpose of hardening the body against colds. But many are not fit for the purpose of hardening; they do not react properly and, instead of feeling refreshed and invigorated after a cold bath, their extremities are livid, benumbed, chilled, and they feel altogether miserable. These patients, independent of their physical condition, are better off when taking only warm baths twice or thrice weekly followed by frictions. The statements of some that every tuberculous patient can be subjected to a process of hardening, if methodically applied, does not hold as is evident from the fact that it is not pursued systematically in most sanatoriums. S. A. Knopf<sup>1</sup> is "convinced that in phthisical patients the routine application of cold water in the form of douch or rain bath can be productive of a great deal of harm." Bed-ridden patients may be sponged with tepid or even cold water during febrile attacks with great benefit. Patients who have been in the habit of taking cold baths, douches or sponging every morning should continue to do so during their illness, but those who do not bear these procedures well should only bathe in warm water, as was just stated.

<sup>1</sup> Medical Record, 1915, lxxxviii, 173.

Robust patients may also be allowed swimming within reasonable limits; bathing outdoors, especially sea bathing, is good for quiescent cases. Turkish and Russian baths are decidedly harmful in active cases.

**Clothing.**—The tuberculous patient should be sensibly clothed; the aim being to keep him warm during the cold winter, but not overheated. The fear for “colds” is responsible for the excessive underwear which we often find on patients, and coupled with the several vests, sweaters, coats, and overcoats, they are often fairly borne down by the weight of their clothing. The well-known red flannel pad, “the chest protector,” has not yet been abandoned after all the medical agitation against it; we often see patients wear them and every drug store sells them. Not only do the poor and ostensibly ignorant classes make use of them, but we meet them among so-called educated patients. They become habituated to this excessive covering of the chest, and perspire freely. When they attempt to remove it they are easily chilled, which is responsible for many of the catarrhal complications which occur during the course of the disease.

In the beginning of the treatment, the patient is to be discouraged from such practices. He is to be told with due emphasis that woolen underwear, of thickness consistent with the season of the year and other meteorological conditions, is all that is necessary. A woolen garment has a capacity for absorbing considerable moisture without feeling wet, while cotton soon becomes saturated with moisture. If evaporation takes place suddenly, the body is chilled. Some patients are unduly irritated by wool next to the skin, but by constant wear they overcome this difficulty. Of course it is important that the underwear worn during the day should not be worn during the night.

All sudden changes in temperature within and out of the house are to be met by changing the overgarments. During the winter a fur coat is good, and can be purchased for about the same price as a good overcoat. Those taking outdoor treatment on a reclining chair need extra wraps during the winter. Carrington<sup>1</sup> gives a complete description of the various appliances which may be used for the purpose.

Women are less easily managed in regard to clothing than men. The low cut around the neck and chest is very harmful to tuberculous women, and they are to be induced to forego some of the fashions in vogue. But what is of most importance is the corset, which many refuse to part with, claiming that it is not at all the figure they care for, but that they have been habituated to stays and feel uncomfortable without them. But when explained in detail the way a corset, even of those called “hygienic,” interferes with the respiratory movements of the thorax, most women submit to the argument.

**Smoking.**—The problem whether a patient who has been found tuberculous should give up smoking has troubled many physicians in sanatoriums. Some have been inclined to prohibit it indiscrimi-

<sup>1</sup> Journal of Outdoor Life, 1912, ix, 262.

nately, and failed, as a rule. One who has been habituated for long years to smoking cannot easily give it up and when he does he is often so nervous and miserable that it has an immense influence on his general well being and the course of the disease. The fact is that smoking has no deleterious influence on the tuberculous process in the lungs, and there is no reason for imposing an additional hardship on the patient. Of course chewing tobacco should be prohibited.

When there are laryngeal complications smoking is apt to cause irritation and cough. However, I am inclined to follow Fetterolf's<sup>1</sup> suggestion: The patient, if he craves for his cigar, cigarette or pipe, is thus instructed: "The smoke is not to be blown through the nose or inhaled; that if a cigar or cigarette is used it shall be smoked in a holder at least four inches long, and, finally that the smoking be done in the open air. The main evils, barring excess, are dry heat and dust which are drawn into the pharynx and larynx. This is of greater significance the shorter the smoked article grows, and if the cigar or cigarette is used in a holder and only the first half is smoked, this evil is largely done away with." It is Fetterolf's belief that with such precautions as just mentioned and with the smoking done in the open air, no harm will result. A non-smoking patient in a close room with others smoking is at a greater disadvantage than one who is smoking in the fresh air.

**Occupation.**—A great deal has been said of occupations fit for tuberculous patients. The problem is not one which concerns those with active disease, but the convalescents, as well as those who have recovered. A patient during the active course of phthisis in any stage should have no occupation at all. He cannot work, he must not attend to any vocation which requires physical or mental exertion. Mistakes are often made in permitting patients in the incipient stages to wind up their business, to finish a course in a school, etc. This is a point which will be discussed later on while speaking on rest and exercise and cannot be emphasized too strongly.

It is very difficult to advise patients who have recovered from phthisis as to their future activities in the affairs of life. With the rich and prosperous the matter is very simple: They may be allowed to return to their vocations provided they know how to take care of themselves. Under supervision, and with careful observation of the ordinary rules of healthy life, they very often avoid relapses. The same is true of professional people who can resume their life work, perhaps at a slower pace. But with those who have been artisans, manual laborers, etc., especially in "precarious occupations," the matter is different. It is, indeed, easy to advise one to change his vocation, as is done in sanatoriums when patients are discharged, but whether the patient is more harmed by working at his trade and earning for his support than by starvation because of lack of funds to buy food, pay for his lodging, etc., is hard to decide.

<sup>1</sup> Hare's Modern Treatment, ii, 405.

Moreover, a change of occupation is not feasible in the vast majority of cases, especially with skilled artisans. They cannot easily accept low wages when at their own trade the pay is much higher and the hours shorter. It is also a fact, only rarely considered by medical men, that the artisan has usually adapted his organism to his peculiar occupation; in fact there is a process of selection going on, certain persons are attracted to certain trades at which they succeed. They must return to these occupations after recovering from the disease, if they are at all to be able to support themselves. And they do, in fact, in spite of our protestations.

But we must try to keep convalescing tuberculous patients from hard muscular exertion, if relapses are to be avoided. They are to be under medical supervision for several months after beginning to work and if they show any signs of damage to their constitution, especially fever, dyspnea, tachycardia, etc., they must stop before it is too late. Nor should a cured patient be allowed to work at any dusty trade, such as pottery and earthenware manufacture, cutlery and file making, certain departments of glass making, copper, iron, lead and steel manufacture, stone cutting, textile trades, fur- or cigar-making, iron-grinding, etc. We have seen the effects of organic, mineral and metallic dust in the direction of engendering a soil susceptible to phthisis. When we bear in mind that a patient with cured tuberculosis almost always harbors virulent tubercle bacilli in the cicatrized area of the lung, we can easily understand that irritating dust may at any time flare up a dormant lesion into renewed activity or cause metastasis.

OCCUPATIONS FOR ARRESTED CASES OF TUBERCULOSIS.  
(W. J. VOGELER.)

Healthy.		Unhealthy.		
Healthy.	Comparatively healthy.	A Because of occupation.	B Factors connected with occupation.	C To employer, etc.
Artificial flower maker	Attendant in insane asylum	Brakeman	Brewery hand	Child's nurse
Banker	Bowling-alley attendant	Bridge builder	Dyer	Baker
Barber		Caisson worker	Emery-wheel worker	Hairdresser
Bone-carver	Boxmaker	Canvasser	Garage	Fish cleaner
Bookbinder	Braider	Car conductor	Gasworks employe	Grocer
Bookkeeper	Brass worker	Cigarmaker	Ice-cream vender	Ice-cream vender
Bootblack	Bricklayer	Coalyard employe	Iceman	Iceman
Broker	Brickmaker	Collector	Glassblower employe	Ice manufacturer
Broom-maker (broom and brush maker)	Cap maker	Constable	Laboratory employe	Milkman
Business man (merchant and dealer, retail and wholesale)	Carpenter	Courier	Marble worker	Nurse
Butler	Carriage maker	Driver	Stone-cutter	Midwife
Buttonhole maker	Cementer	Drayman	Miner	Oysterman
Cabinet-maker	Chemist	Horseman	Pool-room attendant	Seamstress
Chair-caner	Electrical worker	Teamster	Printer	Butcher
Chambermaid	Elevator employe	Engineer	Rag-sorter	Candy-maker
Clergyman	Fireman (fireman and engineer)	Expressman	Reporter	Cook
Clerk (clerk and copyist)	Gasfitter	Farmer	Riveter	Druggist
Cloth examiner	Glazier	Hostler	Sailor	Spice-room worker
Cooper	Gold preparer	Huckster	Scissors-grinder	
Coppersmith (copper worker)	Harness maker (saddle maker and repairer)	Inspector	Stage hand	
Cutter	Houseworker	Iron worker	Stone-cutter	
	Lamp cleaner	Janitor	Type-polisher	
	Laundry worker (male and female)	Junk dealer	Typesetter	
		Letter carrier	Woolsorter	
		Lineman	Wine dealer	
		Longshoreman	Detective	
		Lumber-yard employe		



OCCUPATIONS FOR ARRESTED CASES OF TUBERCULOSIS—Continued.

Healthy.		Unhealthy.		
Healthy:	Comparatively healthy.	A Because of occupation.	B Factors connected with occupation.	C To employer, etc.
Decorator	Masseur	Messenger boy	Hotel and board-	
Designer (archi-	Mechanic	Motorman	ing-house keep-	
and draughts-	Mill hand	Mover	ers	
man)	Molder	Musician	Saloon and resta-	
Dressmaker	Oilworks em-	Navy employee	urant keepers	
Engraver	ployee	Newspaper vender	Livery stable	
Embroiderer	Operator	Painter	keepers	
Factory hand	Packer	Peddler	Tobacco workers	
Foreman (mill)	Paperhanger	Plumber		
Gardener	Penmaker	Policeman		
Hatter (hat and	Pipe-cutter	Watchman		
capmaker)	Plasterer	Porter		
Jeweler	Plaster-of-Paris	Rigger		
Labeler	worker	Salesman		
Labor boss	Rubber-maker	Saleswoman		
Laborer (labor	Sawyer	Scrubber		
not specified)	Seamstress	Shipper		
Lawyer	Statue-painter	Shipwright		
Leather worker	Steamfitter	Signalman		
(currier and	Stereotyper	Soldier		
tanner)	Terra-cotta	Steel worker		
Librarian	worker	Stevadore		
Lithographer	Tin-roofer	Stoker		
Locksmith	Trunkmaker	Street-cleaner		
Machinist	Waiter	Street-paver		
Merchants and	Washerwoman	Tool-sharpener		
dealers	Wheelwright	Undertaker		
Metal worker	Chicken-farming	Veterinarian		
Milliner	.....	Window-cleaner		
Morocco finisher	.....	Wood-chopper		
Nickel-plater	.....	Lumberman		
Office-boy	.....	Raftsmen		
Officials of com-	.....	Miller		
pany	.....	Auctioneer		
Oilcloth worker	.....	Vine-grower		
Optician	.....	Compositor		
Photographer	.....	Pressman		
Physicians and	.....	Newspaper work		
surgeons				
Picture-frame				
maker				
Presser				
Servant				
School-child				
Shirtmaker (shirt				
and collar and				
cuff maker)				
Shoemaker				
Springmaker				
Stand-keeper				
Stenographer				
(stenographer				
and typewriter)				
Storekeeper's em-				
ployee				
Student				
Suspender maker				
Tailor				
Teacher (teacher				
and professor				
in college)				
Telegraph opera-				
tor (telephone				
and telegraph)				
Telephone opera-				
tor (telegraph				
and telephone)				
Time-keeper				
Tin-plater (tin-				
plate and tin-				
ware worker)				
Tinsmith				
Truss-maker				
Upholsterer				
Violin-maker				
Watchmaker				
Weaver				
Woodworker				
Wrapper				

Special efforts should be made to find outdoor employment for patients cured from tuberculosis. It is always to be remembered that farming is not the only outdoor work, nor is it the best. Farm laborers usually work very hard for long hours, small pay and with food that does not satisfy the city dweller. In addition, as has been pointed out by Vogeler,<sup>1</sup> the lack of amusement during the hours of recreation, and the enervating heat during the summer are serious drawbacks. Of course it is different when the patient can raise funds to buy or lease a farm for himself.

There are in cities many more or less remunerative occupations which are suitable for this class of cases, as conductors, motormen, ticket agents, attendants at ferries, watchmen, solicitors, etc. My observations lead me to the conviction that workers at the garment industries, excepting at fur, may safely return to their occupations, provided they find employment in light and well-ventilated workshops. The same is true of the building industry, provided the exposure to the vicissitudes of the weather is not excessive, nor the hours too long; and of clerks, salespersons, etc. Indeed, I have been struck with the fact that when a patient who recovered from phthisis is unable to pursue the vocation for which he has been trained for many years, he will not do well, even if he remains idle indefinitely.

The list of occupations, compiled by Dr. W. J. Vogeler, and reproduced on p. 468, may be consulted when considering a suitable occupation for a convalescing or cured patient.

In judging a patient with a view of selecting an occupation for him, we may be guided by the condition of his temperature, pulse, respiration, and general constitution, but the extent of the lesion is a hazardous criterion. All who have had experience agree with H. M. King that "it frequently happens that a satisfactory condition of health as determined by restoration of working efficiency maintained for many years is not incompatible with physical signs which of themselves would indicate active disease." I have seen many cases in which the reverse was true, the patient showed no signs of active disease in the lung, yet as soon as he began to work he broke down with fever, rapid pulse, dyspnea, etc. These patients cannot work at all. Then there are others who will work for several months and, owing to an evanescent, acute, or subacute exacerbation, are laid up for several days or weeks. With these it is very difficult to judge the ability to work. All tuberculous patients, even after completely recovering from the disease, find it difficult to compete with healthy persons, but the class just mentioned is more apt to lose in the struggle for existence. They must find for themselves employment of a nature which makes them independent of strict regularity.

On the whole, it appears that cured patients do best when returning to their old vocation, for which they have been trained, and at which they can earn the most with the least possible effort. It may be said that, with some striking exceptions, if a patient is not able to pursue his former line of work, he is altogether disabled.

<sup>1</sup> National Assn. Study and Prev. Tuberc., 1912, viii, 113.

## CHAPTER XXXI.

### THE REST CURE.

**Principles of the Rest Cure.**—We know that nature makes a strong effort at repairing the affected lung in tuberculosis, but we only rarely think of the method it pursues when doing it. Examining the chest of a tuberculous patient, we find on inspection that there is a strong tendency to putting the affected area of the lung at rest. As has already been shown, during the early stage the muscles overlying the pulmonary lesion are almost invariably rigidly and spasmodically contracted. This contraction has been ascribed by Rubel<sup>1</sup> to the physiological coördination of the respiratory centre. It inhibits or prevents the motion of the underlying lung to a certain extent. Later, pleural adhesions are formed which impede the respiratory movements of the lung to a yet greater extent, as is seen in the lagging of the affected side of the chest, offering favorable conditions for cicatrization. This immobilization of the affected part of the lung also slows the circulation of blood and lymph in that area, retains the bacteria and their toxic products, thus lessening toxemia and preventing metastatic auto-infection of unaffected parts of the lung. Rubel has shown experimentally that functional rest greatly contributes toward a cure of tuberculous lesions in the lung. He immobilized one lung in rabbits and then infected them by the intravenous way. In the relatively immobilized lung, the lesion was found to be of the chronic and favorable variety, while in the freely movable lung it was acute and progressive.

Surgeons have utilized physiological and functional rest in the treatment of tuberculosis of bones and joints. The modern treatment of Pott's disease and diseases of the various joints consists mainly in affording rest to the affected parts. The splint has done better than the knife in these forms of tuberculosis. Formerly physicians aimed at procuring rest in tuberculous diseases of the thoracic viscera by the application of strips of adhesive plaster, thus immobilizing the thorax; and at present the induction of an artificial pneumothorax puts the affected lung at complete functional rest. "In breathing a normal person 'opens and shuts' the lungs nearly 30,000 times a day," says Webb. "By rest we aim to make the breathing as shallow as possible, imitating almost that of hibernating."

In febrile cases rest has a rationale which is clear to everyone who

<sup>1</sup> Ztschr. f. Tuberkulose, 1908, x, 193, 319; Roussky Vrach, 1907, vi, 648, 721, 750, 896.

gives some thought to the subject. Fever is an indication of activity of the tuberculous process and results from absorption of toxins. By keeping the patient at rest we reduce the frequency and depth of respiration and thus less of the toxins are washed into the blood stream and the fever declines. With the reduction in the fever, there is an amelioration in the cough, and an improvement in the appetite, resulting in better nutrition of the patient.

**Rest and Exercise in Phthisis.**—In former days the treatment of tuberculosis consisted mainly in removing the patient to some country place, or better yet to an institution and urging him to exercise in the open air. Thus, the main principles of the treatment in Brehmer's sanatorium were outdoor exercise for long hours, daily walking, driving, horseback riding, mountain climbing and respiratory exercises. The same methods were followed in institutional and home treatment by many physicians until about twenty years ago.

The development of sanatoriums in which careful observations have been made on the effects of these exercises on tuberculous patients has resulted in swinging the pendulum, and rest has come to the foreground as the most important factor in combating the disease, so that at present vigorous protests are heard from many sides that the indolent life led by sanatorium patients is often more harmful for various reasons than the exercise which was formerly in vogue. Indeed, Paterson reports just as many cures at Frimley where the patients do graduated work, as in sanatoriums in which they are kept at perfect rest for long months or even years.

The contradictory evidence in favor of rest or work is evidently due to the fact that neither rest nor exercise is a panacea which will help in every case, but that each has its indications and contra-indications. When patients presenting symptoms of active and progressive phthisis—fever, anorexia, emaciation, etc.—are urged to work or exercise, considerable harm is often done, and a favorable case may thus be converted into one which is decidedly hopeless. In the later stages of the disease, when the lesion has localized itself, and the patient has no fever, eats well and feels strong enough to do some work, perfect rest may be distinctly harmful, as will be pointed out later on. Rest and exercise have their indications and contra-indications.

**Indications for Rest.**—Nature puts most patients who suffer from active and acute forms of the disease at rest. They are weak, anemic, emaciated, and the exhausting cough, the dyspnea, and the phenomena of toxemia in general, preclude any kind of exercise. But in the chronic cases, or even in some of the subacute cases, the patient may not realize his plight and continue working at his occupation until he breaks down, when it is too late to recoup the lost flesh and forces. Rest, properly applied, in this class of cases may be life saving.

It is clear that all active cases with fever, tachycardia, anorexia emaciation, weakness, etc., are to be kept strictly at rest until most

of these symptoms have disappeared. But it must be stated at the outset that the extent of the lesion is no reliable criterion as to the indications for rest and exercise. A patient in the incipient stage, with a limited and circumscribed small lesion at one apex, and suffering from fever, dyspnea, anorexia, etc., is often more harmed by work or exercises than one in the advanced stages with extensive involvement of both lungs, but with normal pulse and temperature.

With but few exceptions, the rate of the pulse is as good an index of the fitness of the patient to work as there is. As long as it is 90 or over per minute, or it is accelerated to that rate by mild exercises, the prognosis is not good, unless the patient is kept at perfect rest. In tuberculosis we often meet with unstable tachycardia; the pulse runs up to 120 or more per minute at the least exertion or excitement. Such patients are to be kept in bed or on the reclining chair, until we find that mild exercises, like walking slowly on level ground for a half or one mile does not unduly accelerate the pulse. Some of these cases with tachycardia are afebrile, the temperature is in fact very often below normal and exercise may not affect it, but the pulse is accelerated on the least exertion.

Dyspnea, when present, is another sign that the patient must be kept at rest. We must be guarded and not wait for subjective dyspnea, because many tuberculous patients have adapted themselves so well to their difficulties in breathing, that they are not much disturbed by it, and when seen to breathe very superficially and rapidly, even more than thirty times per minute, they may inform us that they suffer no inconvenience in this respect. It is objective dyspnea which should guide us in our estimation of the effects of rest or exercise in tuberculous patients.

Fever has been considered an indication for rest by most writers on the subject; in fact the problems of exercise and rest have usually been solved by the thermometer. In cases of tuberculosis in which the temperature reaches 100° F. the patient is put to bed and kept there till it descends to normal. In acute cases with continuous fever, or during acute exacerbations in chronic cases, or when some complication ensues, such as pleurisy, or any non-tuberculous infection, complete rest is enjoined till the fever abates. In far-advanced cases with hectic fever, reaching a high degree in the afternoon or evening and dropping to normal or even below in the early morning hours, the patient is to be kept in bed at absolute rest. There are, however, cases of tuberculosis with fever which do not require strict rest. They are discussed in detail elsewhere, while speaking of the treatment of fever.

**Technic.**—The rest cure, when indicated, is to be carried out methodically. In acute progressive cases it means complete rest in bed until the temperature declines to below 100° F. Some patients revolt, saying that they feel strong enough to walk around for several hours of the day, that they are lonesome and would surely improve if they were permitted to assume the erect position for some time.

But they are to be told that fever cannot be cured outside of the bed, and as Poujade said: "Undoubtedly prolonged rest in bed weakens a patient, but it weakens less than fever which kills."

In the home of the patient it is advisable, when feasible, to have two beds, in one of which he sleeps during the night, and in the other he spends the day. Considering that the patient may have to remain in bed for weeks or months, the enforced solitude is hard on him, and the change of the bed has some salutary effect. Moreover, these patients are apt to sleep during the day and suffer from insomnia during the night. By changing the room and bed they often become habituated to sleep in one bed and remain awake during the day in the other. One room and bed may also be aired while the other is used.

In the morning, when the patient wakes, he is to be given a sponge bath—one with alcohol is invigorating—and dressed, the lower half of the window opened and the bed placed in such a position that he can look out on the living world. If he feels cold, a hot-water bag may be placed at his feet. Great care must be taken to prevent bed-sores in prolonged and advanced cases.

When the temperature descends below 100° F., or even in prolonged cases when it reaches this degree only at a certain time in the afternoon, but is near normal during the rest of the day, the patient may be kept at rest on a reclining chair during the greater part of the day, preferably outdoors, and reading and mild games may be allowed; only during the hours when the rise in temperature is expected is he to be made to go to bed. When we find that this does not increase the fever, he may be permitted mild exercises, such as short walks, and the effects should be watched. We are often surprised to find that the fever disappears altogether with mild exercises.

This rest in bed is at times very difficult to carry out. The poor are often working for weeks while the temperature is high—I have seen them working with fever of 103° F. and even higher. When beyond control in this regard, the patient is to be sent to an institution, or to one of the day and night camps. I have seen excellent results in such cases after the patient has been at one of these institutions for a few months. Not only has the fever disappeared, but the patient was educated to appreciate the dangers of exercises during the febrile stage. But the well-to-do are not better in this respect. Very often we find them walking around, and even dissipating, in spite of the fact that their temperature is above 102° F. Indeed, they are often less amenable to reason in this respect than the poor. They are to be impressed that all business and pleasures are to be given up when the temperature is high.

**Contra-indications.**—It was one of the great mistakes of many sanatoriums to urge all patients to keep at perfect rest and abstain from work or exercises, irrespective of the form of the disease and the constitutional symptoms. The result was that they turned out lazy

people—hypochondriacs—who feared work and who at the least fatigue considered themselves harmed by it after they had been cured. In most sanatoriums of today strong efforts are being made to avoid such mistakes.

As was already stated, the extent of the lesion is not always an index as to the indications for rest. There are many patients with extensive lesions in the lung, in fact with large excavations, who are well able to make themselves useful along certain lines. In fact, there are cases in which prolonged rest is distinctly harmful. The nervous system may be functionally damaged beyond repair, the desire for activity may be stifled, and the resistance of the body in general may be lowered. It has also been shown by Paterson and Inman that prolonged rest deprives the patient of certain reactions which bodily activity calls forth in the pulmonary lesions and which are of great use in combating the deleterious effects of the disease.

In some sanatoriums where the rest cure has been carried to excess we often meet with patients who after remaining in bed or on the reclining chair for several months, become mentally tired and listless; they lack interest in current affairs; others become hypochondriacs, consulting the thermometer several times a day and are alarmed at each finding above or below normal. They often lose all hope of ever getting cured and this despondency contributes greatly to the unfavorable course of the disease.

The graduates of sanatoriums in which the rest cure is carried to excess are apt to be lazy for the rest of their lives. Some of them, discharged from one institution immediately seek admission to another. As Herman M. Biggs says: "A sick workman is converted into a healthy loafer." They fear muscular exercise of any kind and imagine that the least work aggravates their condition. In the State and municipal institutions in this country we find many with a record of having been in several sanatoriums. In fact, prolonged rest disables any human being, because the muscles become stiff and any attempt to walk produces muscular weakness, pains and aches in the limbs. In some, the long rest favors the deposition of fat, which is very encouraging, but when carried to excess, which is not a very rare phenomenon among the tuberculous, it may disable the patient as much as active phthisis. These patients must have exercises to reduce the fat. This is mainly seen in patients in whom the disease may or may not be active, but at any rate is not progressive; the lesion has become quiescent, completely surrounded by connective tissue. Rest may only produce obesity of various degrees, but does not assist in the healing of the disease focus in the lung. It is in these cases that graduated work or any exercise will do more than rest, and McLean's aphorism "if the phthisical patient would live, he must work for it," is confirmed.

**Exercise.**—When the temperature and pulse become normal and remain so for several days, walking exercises are to be commenced,

with a view of preventing the deleterious effects of idleness as well as provoking mild reactions—auto-inoculations, which are, in most cases, of immense benefit. At first the patient is allowed to walk a mile on level ground and the effects on the temperature and pulse are watched. It may be done during the morning hours, when the temperature is normal, while in the afternoon, when there is some fever, the patient is ordered to rest on a reclining chair, or even in bed. But in those in whom the afternoon temperature is mild, below 99° F., even this precaution need not be taken, provided the pulse is below 85 per minute.

The following schedule for walking exercises, modified after that given by E. Hyslop Thomson,<sup>1</sup> may guide the patient who takes his own temperature:

Morning temperature at 7 A.M.	{	98.5 or lower; long or medium walk. 99.0; short walk. 99.5; rest outdoors or short walk around house. 100.0 or higher; remain in bed.
Temperature at noon	{	99.0 or lower; medium or short walk. 99.5; short walk. 100.0 or higher; rest in bed or reclining chair.
Evening temperature at 7 P.M.	{	99.5; only short walk on the following day. 100.0 and above; complete rest during following day.

Hill climbing, or walking long distances, up to fifteen miles a day in afebrile cases without tachycardia may be permitted. The author has thus tested patients as to their ability to work, and was surprised to find often that they were rather invigorated by the exercise and they were then allowed to work for their support. Our patients are told to come to the office on foot, walking a mile or two, and if when they arrive the pulse and temperature are found normal, they are told to walk a longer distance the next day, etc. When this test shows that no harm is done by the exercises the patients are allowed to work, first under supervision, and later completely discharged with instructions as to the signs of danger.

**Graduated Labor.**—Practitioners among people in large cities are often impressed with the capacity for work of many consumptives amid unfavorable surroundings for years without visible harm. Among these cases there are many who are evidently active but not progressive; some are entirely quiescent. We must repeat that the extent of the lesion is less of an index as to the capacity for work than its activity as revealed by the constitutional symptoms, such as fever, tachycardia, dyspnea, etc. Paterson<sup>2</sup> developed his system of graduated labor after observing such cases in England. "It occurred to me," he says, "that if some consumptive persons under adverse circumstances, and without any medical guidance, could act thus without

<sup>1</sup> Consumption in General Practice, London, 1912, p. 223.

<sup>2</sup> Sixth Intern. Congr. Tuberc., 1908, i, 886.



apparent injury, they ought, under ideal conditions and with the work carefully graduated in accordance with their physical state, to be able to undertake useful labor. On this assumption manual work should be of great advantage to patients undergoing treatment in a sanatorium, as, at first, it would do much to meet the objection that members of the working classes are liable to have their energy sapped, and to acquire lazy habits by such treatment; second, it would make them more resistant to the disease by improving their physical condition; and third, would enable them by its effects upon their muscles, to return to their work immediately after their discharge."

With a view to developing the muscles of the upper limbs, which are supposed to have more direct influence on the expansion of the lungs, Paterson is not satisfied with walking alone. When a patient is found to be able to walk ten miles a day without aggravating his condition, he is given a basket in which to carry mold for spreading on the lawns, etc. No case of hemoptysis or of pyrexia occurred among these patients. When they had been on this grade with nothing but beneficial results for from three weeks to a month, they are given boys' spades with which to dig for five minutes, followed by an interval of five minutes for a rest. After a few weeks, several of the patients on this work, who were doing well, were allowed to work as hard as possible with their small spades without any intervals of rest. As they had all improved on this labor, larger shovels were obtained, and it was found that these patients were able to use them without the occurrence of hemoptysis or of a rise in temperature. About this time many of the patients were feeling so well that it became necessary to restrain them from doing too much.

Paterson worked out a schedule for graded work which brought excellent results. It was noted that many patients on their arrival are somewhat remarkable for a somewhat sullen and apathetic attitude, but as soon as their physical condition undergoes amelioration, all traces of gloom and depression leave them and they become lively, cheerful individuals. In many cases in which the improvement was not prompt, the effect of harder work was tried and often a progressive improvement was noted at once. Paterson found that the danger signals are: a temperature of 99° F. or higher in men and 99.6° F. in women, loss of appetite and slight headache. As soon as these appear the patient is to be put to bed till the temperature goes down to normal. In my experience, a rapid pulse is of even more importance as an indication that exercises are deleterious.

Inman,<sup>1</sup> working with Wright's method of ascertaining the opsonic index in patients under this graded work system of Paterson, found that it was at some part of the day well above normal and he explains it as due to the stimulus supplied by the work, inducing artificial auto-inoculation; that the organism responds by the the production

of immune bodies. In fact, whenever excessive auto-inoculation takes place harm is done. This, he points out, must be readily recognized clinically, if harm is to be avoided. "A patient doing well on the grade of work prescribed for him and with no abnormality of temperature suddenly complains of feeling tired, of loss of appetite and of headache; and the temperature chart registers an elevation to 99° or 100° F. These are precisely the symptoms which are found during the negative phase after excessive dose of bacterial vaccine."

Paterson is guided in his conduct of a case by the thermometer, and whenever the temperature registers 99° and over in men and 99.6° in women (by mouth), the patient is kept strictly in bed. When work has been assigned, the temperature is watched, and as long as it is not increased by the exertion, the work is increased in duration and intensity. Even afebrile patients who are of poor general condition are not allowed to work, but kept at perfect rest, excepting that they are allowed to walk to and from the dining hall for their meals.

It is thus evident that there is little new in this system of exercises and work. Physicians have always allowed their afebrile patients who are of good general condition and not easily fatigued to work and warned them to stop as soon as symptoms of toxemia, such as a tired feeling, weakness, debility, drowsiness make their appearance. Intelligent patients have been given thermometers to aid them.

Paterson's method has, however, done a great deal for institutional patients by drawing attention to the importance of exercises and work in attempts at prevention of indolence which, in many cases, remains as a reminder of the disease and the institutional life to which they had been subjected.

**Outdoor Games.**—Afebrile patients without tachycardia are to be encouraged to do some exercise in the open air, otherwise they are likely to brood over their troubles, and in some cases even harmed by obesity. Walking exercises alone are often insufficient to keep the average patient busy, and outdoor games are often good to help him pass this time pleasantly and to benefit the muscles, the appetite, and the metabolism.

In advising a patient as to outdoor games we must always consider his life, habits and customs before he took sick. Those who indulged in sports may be permitted to resume their favorite games, provided they do not raise the temperature or produce breathlessness. This at once excludes certain games. "All violent sports should be avoided," says Lawrason Brown, "Golf (without the full swing), croquet, fishing and hunting (not entailing too much exercise), gentle bicycle riding (on the level), rowing or paddling, skating (for those proficient), skiing, snow shoeing, swimming (in great moderation), and horseback riding, may be indulged in with moderation when the disease has been arrested."

It seems to me that of the outdoor games, golf is the best for patients who have just recovered from phthisis. Cricket, football and athletic

sports in general produce more or less dyspnea, while golf makes less violent demands on its votaries, and is usually played in open breezy places.

**Indoor Games.**—The tuberculous patient is to be allowed some games for his amusement even when he is kept indoors, excepting when the temperature is above 100° F. and he is kept in bed during the whole day. I believe it is wrong to interfere with them when they play cards, checkers and chess, as is often done in public sanatoriums, on the assumption that the excitement is liable to raise the temperature, provoke hemoptysis, etc. While it cannot be said that these games immunize the patients against such accidents, I have never seen such results follow when they are allowed to have some amusement during the long, lonesome days and weeks in the institution.

Patients treated at home are not to be allowed to go to theatres, or other indoor and badly ventillated places of amusement as long as they have fever.

## CHAPTER XXXII.

### OPEN-AIR TREATMENT.

Most writers state that Brehmer was the first to demonstrate, in 1859 in his sanatorium, the great value of the open-air method of treatment of tuberculosis. But it is a fact that he had many precursors. In 1840 George Bodington, a country doctor in the village of Erdington, published an *Essay on the Treatment and Cure of Pulmonary Tuberculosis*, in which he vigorously protested against the close confinement of consumptives for fear of the evil influences of cold, fresh air, "forcing them to breathe over and over again the same foul air contaminated with diseased effluvia of their own persons." Arguing against the value of antimony, calomel and bleeding, which were in vogue in those days, he urged the free administration of nutritious food and stimulants with plenty of exercise in pure and, if possible, dry, "frosty" air. In short, his great specific in phthisis was dry, cold air which, he said, had a most powerful influence in "healing and closing of cavities and ulcers of the lungs."

Needless to say he was severely handled by his contemporaries and so discouraged that he had to give up his method of treatment, converting his "sanatorium" into an insane asylum. Brehmer in Germany and Trudeau in the United States later took up work along the lines of Bodington and met with no small amount of opposition and ridicule from the contemporary leaders of the profession and the laity.

At present the gospel of fresh air needs no evangelists to bring it home to most sufferers from phthisis; it is the acknowledged cornerstone of phthisiotherapy. The only difference of opinion is where and how it can be applied most effectively. Some send their patients to certain regions where the climate is alleged to have a specific influence on the disease; others direct them to sanatoriums where they may benefit by both the climatic advantages and certain therapeutic methods which are the hobby of the presiding genius. Many are convinced that similar advantages may be obtained at the home of the average patient.

**Where Open-air Treatment may be Obtained.**—The open-air treatment consists in inducing the patient to live permanently in pure, fresh air, preferably outdoors or, when he must stay indoors, the air in the room is to be renewed constantly. There is no question but that this is best obtained in the country or in a special institution. But most patients cannot afford to leave the city for an indefinite period, nor are there a sufficient number of institutions in any country to accommodate all active tuberculous patients with places for as long

as the disease lasts. In fact, if all the patients were to decide that they want to submit to hospitalization for therapeutic or prophylactic purposes, it would be found that only a small fraction of the eligible could be accommodated.

Says Edward Cummings:<sup>1</sup> "Personally I cannot see the need of banishing the tuberculous patient from his comfortable chamber to a shack in the back yard, or a woodshed, or a tent house in the dusty desert. One does not always have to go across the continent to get fresh air, not even out in the yard. . . . The ordinary bedroom for most persons is well enough." My own observations in large modern cities like New York, Boston, Chicago, St. Louis, Philadelphia, London, Manchester, etc., have convinced me that results can be, and are, obtained which compare favorably with climatic and institutional treatment. Of course in the congested districts and slums, the overcrowded tenements are even less suitable for consumptives than they are for human habitation in general. But there are districts in every city which can be utilized for the purpose of housing consumptives and the results attained will not be behind those attained after sending patients far away from their homes at great expense and often hardship. - Dr. Thomas Spees Carrington has done a great deal in popularizing suitable methods of open-air treatment for consumptives in cities.

The suburbs around cities are suitable for families in which there are tuberculous members and the expense involved in moving them to these parts is comparatively trifling; in fact the rent is often lower, and they need not lose their jobs or break up their business. The social and economic aspects of tuberculosis, which are but rarely considered in this connection, assume a different aspect when the patient must not be sent far away from home or from the place of employment of those he depends on. Lawrason Brown<sup>2</sup> thus summarizes the question: "Treatment at home is less expensive, does not entail residence in a hotel or boarding house or 'acclimatization' after return to work; precludes separation from the family and friends, fatigue of travel, homesickness; does not break up business arrangements in the same degree."

**Open-air vs. Climatic Treatment.**—These two methods must be kept distinctly apart. Experience has taught that there is no climate on the habitable globe in which consumption does not occur, or where a patient sick with the disease will surely recover, even when sent thither in the very incipient stage of the ailment. In the climatic resorts which have become popular—and it is a fact that the popularity of a region is by no means an index of its therapeutic efficacy—the patient must subject himself to a certain discipline if he expects results. Irrational mode of life in the mountains or at the sea coast will aggravate the condition of a consumptive to the same extent as it will in the

<sup>1</sup> Journal of Outdoor Life, 1912, ix, 257.

<sup>2</sup> Osler's Modern Medicine, i, 487.

city. A healthful mode of life in any place will, and does, improve the condition of the average consumptive, no matter where he is.

The treatment of tuberculosis in certain climatic regions, as we shall see later on, has its indications and contra-indications, while home treatment has certain advantages in this regard. It can be applied successfully in the treatment of nearly all cases, in all forms of phthisis, and in all its stages; striking results are obtained in patients with limited means as well as with those who are well-to-do; in febrile and afebrile cases; in hemorrhagic and cachectic cases; in those with or without gastric derangements. In short, in all cases of tuberculosis, in all its forms, in all stages of the disease, during any season of the year in almost any climate, except the arid.

To be successful, it must be applied rigorously, methodically and properly regulated by the physician. This is no more than institutional treatment depends on, excepting that the former is cheaper for the community which is charged with caring for its consumptives and more attractive to many who have sufficient material means at their command.

**Dangers of Stagnant Air.**—Our conception of the beneficial effects of indoor life has undergone radical changes during recent years. The reasons why the stagnant air in a room occupied by human beings is harmful are not clear. Recent investigations by Leonard Hill, Haldane, Benedict, Flügge, C. E. A. Winslow, and others have shown that it is not the excess of carbon dioxide, or the decrease in the proportion of oxygen which renders the stagnant air harmful. The most deteriorated air in a badly ventilated room never shows on analysis as much as 1 per cent. of carbon dioxide, while in famous health resorts at high altitude there is a far greater deficiency of oxygen than can ever be found in the worst ventilated room. The specific organic poisons of human origin, the morbid anthrotoxins, of which some have spoken, have never been isolated.

As Winslow<sup>1</sup> points out, recent studies indicate beyond any reasonable doubt that the more obvious effects experienced in a badly ventilated room are due to the heat and moisture produced by the bodies of the occupants, rather than to the carbon dioxide or other substances given off in the breath. Two fundamental experiments have been repeated again and again which would suffice to demonstrate, as F. S. Lee has so well expressed it, that the problem of ventilation is not chemical but physical—not respiratory, but cutaneous. These are: First, that subjects immured in close chambers and exposed to heat as well as the chemical products formed therein, are not at all relieved by breathing pure outdoor air through a tube; and second, that they are completely relieved by keeping the chamber artificially cool without changing the air at all, and are relieved to a considerable extent by the mere cooling effects of an electric fan.

<sup>1</sup> Science, N. S., 1915, xli, 625.

Investigations made by the New York State Commission on Ventilation have shown that the temperature and the pulse rate of an individual are markedly increased by even a slight increase in the room temperature; they also confirm Leonard Hill's observations that overheated rooms enhance the susceptibility to respiratory diseases owing to changes in the mucous membrane which follow exposure to hot and dry air, and the resistance of animals to artificial infection is very definitely lowered by chill following exposure to a hot atmosphere.

In connection with tuberculosis, in which the appetite is of such great importance, it is of interest that stagnant air reduces the desire for food perceptibly. In two series of experiments made by the above-mentioned Commission, standard luncheons were served to the subjects in the experimental chambers and the amount on their plates weighed. In one series the subjects consumed on the stagnant days an average of 1151 calories and on the fresh-air days an average of 1308 calories, an increase of 13 per cent. In a second series during colder weather, the average consumption was larger, 1492 calories for the stagnant and 1620 calories for the fresh-air days.

We have here an explanation for the utility of fresh air in the treatment of tuberculosis. Stagnant air is bad primarily because of its high temperature and lack of cooling air movement, sometimes combined with high humidity. In fact a lack of humidity, as Phelps has pointed out, makes hot air feel cooler and cold air feel warmer. It is very important that the air in a living room should not be dry, as it is in most of our artificially heated rooms during the winter. W. Freudenthal<sup>1</sup> has shown the dangers of dry air in a recent study of the subject. Living in stagnant air the patient feels uncomfortable, inert and listless, and above all, loses his appetite for food, which is very essential in the treatment of phthisis. The open-air treatment seeks to remove the drawbacks of indoor life amid stagnant air. No doubt it is attained best in a good sanatorium, but it may be just as well attained at home within the city lines in most houses.

**Technic of Treatment.**—If the patient lives in a capacious home, or in one in which he may have a fair-sized, well-lighted, and ventilated room to himself, in a district or street which is not overcrowded, he may remain where he is. But in case he lives in the slum district of a large city, in a dingy and overcrowded tenement, he must move to better quarters which are available in every city. If his occupation, or that of those he depends on, is not in the way, it is even better that he move to the outskirts of the city, or to a suburb where certain advantages may be obtained which are not available or feasible in the city.

A few words should be said about the various shacks, tents, special window tents, etc., which have been contrived for the city dweller with a view of giving him an opportunity to live outdoors or in a well-

<sup>1</sup> New York Med. Jour., 1914, xcix, 1.

ventilated room. Most of them are not feasible. They cannot be used in the thickly inhabited parts of cities; the tents or shacks cannot be placed in the back yards, on the roofs, etc., without attracting the curious, or even exposing the patient to eviction because of the resentment of the neighbors. I have seen a few patients in New York City who have made use of these contrivances, but they were rare exceptions, and they lived in private dwellings in the outskirts of the city.

But the average bedroom, excepting in the dingy tenements, is sufficient for our purposes. If the patient is allowed to remove the window sashes, both the upper and the lower, as Cummings suggested, he may convert it into an open-air sleeping quarters. The patient's room should be large, one with a capacity of 3000 to 3500 cubic feet of air is best. But it must always be remembered that cubic space is of little value *per se* unless it is provided with efficient means of ventilation.

In modern apartments, rooms with windows opening into air shafts or narrow courts are not good for tuberculous patients; they should have rooms with windows opening into the street or a spacious courtyard. In apartment houses with elevators the top floor is the best, the higher the building the better. But in houses without elevators the advantages of the pure air in the upper stories are often negated by the exertion necessary in stair climbing by walking patients; but the ground floor should be avoided. It should also be seen that trees do not obstruct the entry of air and light to the room and favor excessive humidity. The windows of the room must be located so that the sun's rays enter them for at least part of the day and penetrate at least ten feet into the room.

The walls of the room should be painted, not papered. All unnecessary curtains and hangings should be discarded, leaving nothing but roller shades on the windows. Carpets are obviously bad, but some rugs should be left on the floor. Bare floors are apt to discourage the patient as well as those around him. The rugs can be taken out at frequent intervals, aired, and disinfected. The floor should be waxed or painted, so as to be easily cleaned. Steam or hot-water heating is best; gas heating is to be avoided because it consumes oxygen from the air.

Afebrile patients who are allowed outdoor exercises, should remain in the room very little during the day. In the city they are to leave their room soon after breakfast and go to some neighboring park where they are to spend the greater part of the day. In the outskirts of the city, or in the suburbs there may be sufficient space around the house, as well as porches, balconies, etc., on which they may exercise and rest comfortably, reading or doing some light work under careful supervision of the physician. Intelligent patients may be given thermometers with directions to guide them as to the effects of exercise or work.



The season of the year has little effect on the outdoor life. The patient is to spend the greater part of the day outdoors during the winter as well as during the summer. Only intense cold, or sun rays, rain or strong winds are to be avoided by seeking shelter. Excepting during blizzards, snow is rather invigorating to the average patient of this class.

**Sleeping Porches.**—Those living in the outskirts of the city or the suburbs, may have tents in which they sleep during the night and seek shelter during inclemencies of the weather. But the usual tent is rather stuffy and damp for a tuberculous patient. There are made at present tent houses, or canvas bungalows, which are excellent because of the comforts they afford and the good ventilation that may be had within them.

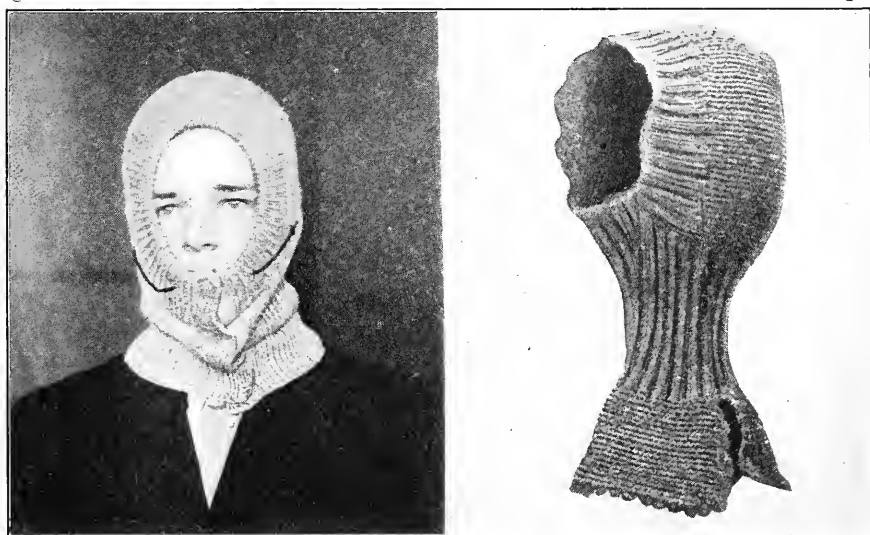


FIG. 80.—A knitted helmet for protecting the head, neck, and shoulders. (T. S. Carrington.)

It is, however, best that the patient remain the greater part of the day on the porch, and in most cases he may sleep in a bed placed on the porch. During the day, in case perfect rest is to be enjoined, he may remain on some form of reclining chair of which there are at present many on the market, such as the Adirondack Recliner, the Kalamazoo Chair, the common hammock chair, the willow long chair, etc. During the cold winter he may also remain on the porch on one of these chairs during the day, and in a bed during the night. "The whole problem is one of sufficient bedclothes and the use of some sort of hood or head covering (Fig. 80); in short, to dress especially for sleeping out." As Cummings suggests, "by putting on a suit of underwear, a flannel shirt, pajamas of outing flannel, and a hood of flannel

or eider-down, and furnishing the bed with plenty of light weight, but warm blankets and comfortables one can sleep with a continuous flood of fresh air in severe weather and perfect comfort and safety."

It is self-evident that sleeping porches are only feasible in rural districts, and not in large cities, excepting in their outskirts. But it is always important to remember that the proper construction of a sleeping porch is not a simple matter. A. Morgan MacWhinnie<sup>1</sup> investi-

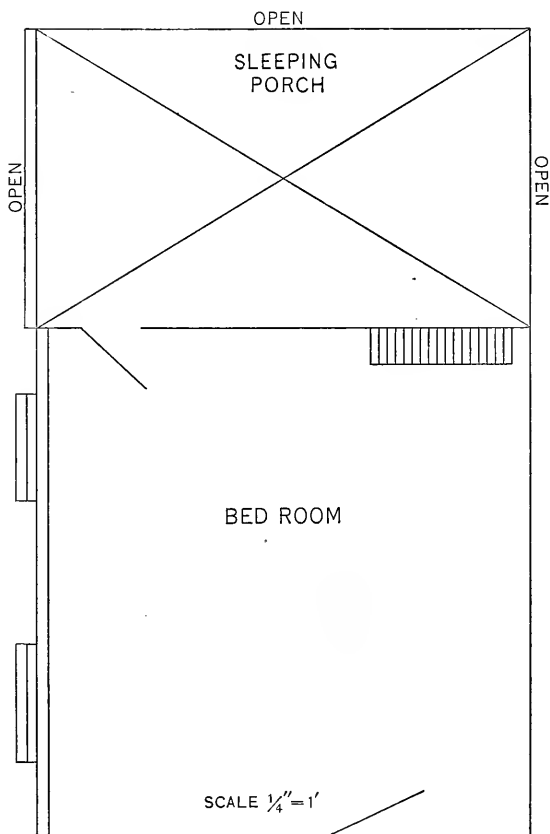


FIG. 81.—Porch exposed on three sides; no provision for keeping the bed warm during the day. (MacWhinnie.)

gated one hundred sleeping porches in the Northwest and found the following conditions: In 96 cases the sides of the sleeping balcony were partially protected from the wind and rain by a tarpaulin or some other material. Two had no protection whatever, and one was inclosed with glass windows which could be thrown open horizontally at night on retiring. This was the only one that could be closed in

<sup>1</sup> New York Med. Jour., 1914, xcix, 780.

the daytime, and had hot-water radiators connecting with the boiler in the cellar that kept the bed and its covering as warm all day as the rest of the house. In 98 cases the bed, mattresses, linen, and covers were exposed all day to the dampness of the atmosphere. I found similar conditions in most of sleeping porches in the East.

The warming of the bedding and coverings, and keeping them dry is an element which is very often neglected in open-air treatment and it is not surprising that most patients do not want to sleep outdoors on cold and moist days. MacWhinnie suggested sleeping porches

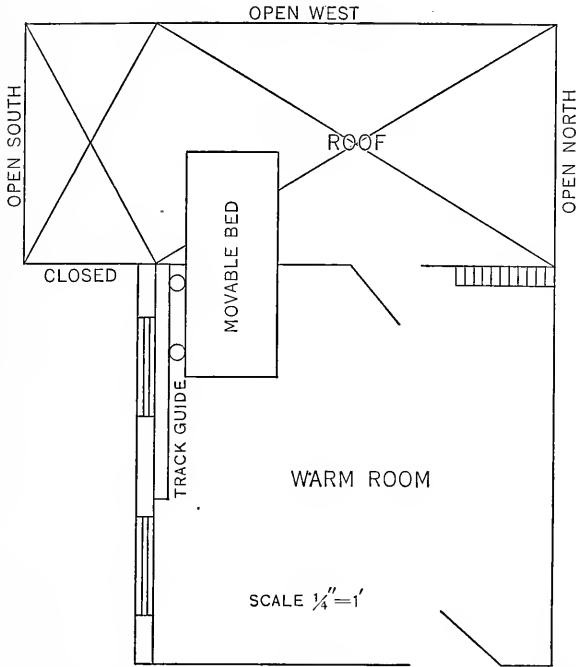


FIG. 82.—Ideal sleeping porch. When the bed is fully extended on the porch, the footboard closes the room from the outside air; when bed is in warm room, headboard closes opening to sleeping porch. (MacWhinnie.)

which have none of these disadvantages; they are so arranged as to be completely protected from the weather. He urges that the doors should be large so that the bed can be kept in the heated room during the entire day so that the bedding is warm and dry. When ready for the night, it should be wheeled onto the sleeping porch, thus obviating the disadvantageous conditions mentioned above.

Figs. 81 and 82 show the plan of a sleeping porch, designed and constructed by Dr. D. C. Hall. An opening is made in the wall large enough for the bed to roll through onto the porch. The head and foot boards are so constructed that the opening in the wall is entirely

closed when the bed is at full length on the porch or in the room. The room is thus kept warm for dressing in the morning. The bed is supported by four large roller bearing wheels, one hand of a child sufficing to move it out or in. Grips are so arranged that the bed can be drawn out or in, while the occupant is in the reclining position.

**Open-air Treatment of Febrile Patients.**—The afebrile patient may indulge in driving, automobiling, or sleighing during the winter, but always within the limits set by the physician.

He should discard many of the pleasures of healthy people, even when he thinks he is well; he should not visit theatres, balls, crowded restaurants, etc., where large numbers of persons congregate and contaminate the air. Many a patient who has been doing well, and was on the road to recovery, has suffered a relapse or a complication, after attending a function at which a large number of persons got together in a confined space.

With febrile cases things are not so simple. They must remain in bed as long as the fever lasts, excepting under circumstances which are discussed elsewhere. In the city the bed can only be kept within the room and for this reason, as well as for others, it must be placed near the window, so that not only pure, fresh air may be available at all times, but also because the patient is usually encouraged looking out at the living world. In the suburbs the bed may be placed on the porch during the day, and under certain circumstances it may remain there all the time. When feasible, a proper tent or porch is even better. Placing tents on roofs of houses in the city, or modifying fire escapes so that the patient may be kept on them in the open air, are not feasible. No patient wishes to expose himself to the curious-gaze and commiseration of the other inhabitants of the house, as was already mentioned.

The good effects of the open-air treatment are very striking in febrile cases. The general condition of the patient improves, a feeling of well-being ensues, replacing the despondency into which he was sinking. His strength returns. The anorexia and indigestion which sapped his strength disappear, or are ameliorated, and he eats with a better appetite. The painful cough often disappears within a few days and nights with open windows or on the porch. This is at times the most salutary phenomenon; sometimes when sedatives have failed to control the cough, outdoor life works in this direction and the effect on the morale of the patient is marvellous.

We often have patients who, in mortal fear of "colds," decline to carry out the open-air treatment; their relatives and friends discourage them yet more. But several days' experience along the line just described convinces the average patient. At the Montefiore Hospital, where the patients come from the tenement districts of New York City, and have always feared open windows, they soon find out the advantages of fresh air and would strongly resent any attempts at closing the windows. It is often necessary to control the "fresh-air

fiends," when conditions arise which necessitate their remaining indoors for some time.

The superstitious fear for colds and draughts is one of the greatest drawbacks in phthisiotherapy. The patients are apt to ascribe all their troubles to colds. After passing through an acute exacerbation of the disease, which they usually ascribe to a cold; or getting some pain or ache in the chest, or hoarseness due to laryngeal complication, etc., they begin to fear exposure. This is to be discouraged. The patient is to be told clearly and distinctly that his troubles are not due to fresh air, but to the lack of it, and that a cure can only be attained by living outdoors.

During the night the open-air treatment is just as simple as during the day. It consists in one simple principle—open windows. They must be opened completely; the upper half must be completely lowered and the opening should not be obstructed by any shade or curtain. Patients who have not slept in a well-ventilated room—the fact that they are phthisical shows that they have not—and are not habituated to cold air during the night, rebel when told to open their windows widely during winter nights, but a trial of a few nights convinces most of the sceptics.

With obstinate patients we may begin by lowering the windows one-third; after a few nights the opening is increased to one-half, etc., so that within a week or two the patient finds out that sleeping with a free current of air invigorates him and he will not tolerate their closure.

Half-measures, such as opening the windows in adjoining rooms, etc., are not to be tolerated. The patient should be impressed with the fact that it is not only fresh air we are looking for, but a free circulation of it and this can only be attained by keeping the windows open in the room he inhabits.

As a rule, there is no necessity for heating the sleeping room for the night during the greater part of the winter. Warm sleeping rooms are badly ventilated. Only during the very cold days is there a necessity for heat, but the windows must remain open. Careful measurement has shown that the temperature within the room is always above that outside and the humidity is lower. A sufficient number of blankets and plenty of flannel underwear, used according to the temperature, will keep any patient warm. The fear entertained by many patients that exposure of a limb in a cold room may be harmful is not supported by facts observed in daily practice. The human being keeps its limbs instinctively covered when sleeping in a cold room. Moreover, insomnia is sure to occur if he is not well covered. It is also a fact that persons lying in bed well covered feel quite warm in a room so cold that those around find it difficult to bear, as is the experience of nurses attending to outdoor patients.

It is self-understood that very few patients will at once begin the treatment by undressing in a cold room during the winter and going to

bed and again dressing in the morning in a cold room. For this reason it is much easier to institute the treatment during the summer. But in winter we may begin by warming the room an hour or so before the patient is expected to retire and again before he rises in the morning. But in time many patients discover that all this is unnecessary and they undress and dress in a cold room without a murmur. In many cases the patients prefer to have an adjoining room for this purpose.

Wind, rain, and snow are not sufficient reason for closing the windows of the sleeping room of the patient. This must be insisted upon and the patient should be convincingly told that it is the fresh, circulating air which replaces his expired air and cools his body that keeps up his vitality. Even during complications of phthisis the windows are not to be closed in the vast majority of cases; most of these are prevented or cured by fresh, cold air.

In moderate climates consumptives feel better during the winter, as was already shown when discussing hemoptysis, emaciation, etc. It is the universal experience that when the summer heat is accompanied by excessive humidity tuberculous patients suffer from anorexia, insomnia, general weakness, etc., and they often lose the greater part of what they have gained during the cold winter. For this reason I insist that all patients under home treatment should leave at least for the two months of July and August for the mountains. It is also well that during warm days an electric fan should be installed in the rooms inhabited by consumptives for reasons already made clear.

**Results Attained by Open-air Treatment.**—The results attained by the open-air treatment depend on many conditions, notably the acuteness and the stage of the disease. In acute, progressive cases we cannot expect much more than from any other method of treatment, excepting perhaps more comfort to the patient than would be the case if he were kept indoors. The ultimate prognosis is gloomy at all events. In fact, if these patients insist that they cannot bear the cold, it is of no use arguing with them; it is best to let them have their own way during their last earthly days. In subacute cases the process is at times arrested and the disease then pursues the course of chronic phthisis.

The good effects of the open-air treatment are best seen in the average case of incipient chronic phthisis which begins with moderate fever, nightsweats, anorexia, cough, etc. In advanced cases of the disease, when the patient is emaciated and apparently hopeless, several days of life in the open air often transform a despondent individual into one who shows his confidence in ultimate recovery very clearly. He gains in courage and a desire for recovery; his fever declines, the nightsweats disappear, the cough and expectoration diminish, and he becomes hopeful in general.

In the far-advanced stages of the disease the open-air treatment may only render the last days of life somewhat more bearable, contribute to the false optimism which is often seen in these patients, and

accentuate the euphoria which has been considered characteristic of the disease. But it is undoubtedly curative in the vast majority of incipient cases. The entire aspect of the patient is often transformed within a week or two, and the improvement is usually progressive. A good appetite and assimilation and digestion of the food, disappearance of the fever, nightsweats, insomnia, and amelioration of the cough, are the rule in these cases. Often it will be noted that fever which resisted all other treatment for months disappears after several days of life with open windows during day and night. Many patients learn it by experience and cannot be induced to close the windows. They have found that with open windows they sleep better and feel refreshed in the morning, while closed windows induce cough, nightsweats, insomnia, listlessness, etc.

**Contra-indications.**—It must be emphasized that there are but few contra-indications to the open-air treatment. Even hemoptysis, however severe, should not induce us to close the windows of the room inhabited by a tuberculous patient. Nor should they be closed during any season, as was already mentioned. Only during the summer, when the external air is often hot and humid, and even open windows are not effective in producing a free circulation of the air within the room, this method is often futile. An electric fan may improve conditions somewhat, but it is best that patients who can afford it should leave the city for a milder or colder region.

There is a small number of patients who do not bear the open-air treatment very well during the winter months; in fact in some it is distinctly harmful, and if an attempt is made to apply it, it must be done with great care and circumspection. Patients who suffer from diffuse bronchitis in addition to phthisis do not bear cold air very well and so-called "rheumatic pains" in the joints are often aggravated by sleeping in a cold room. Cold air is also bad for consumptives who suffer from organic heart disease—dyspnea and the cough are decidedly provoked by winds, draughts, and cold air in general. Those suffering from profound anemia at times cannot be kept warm by any means in a cold room. Some nervous patients who have obstinately made up their minds that the cold is harmful are also bad material for this mode of treatment. The same is true of old persons with bad peripheral circulation, and extremely cachectic patients—they cannot be kept comfortable in cold rooms during winter nights.

In all these cases it is necessary to heat the room, but the windows should under no condition be closed completely. On the other hand, when some complication ensues, such as influenza, pleurisy, pneumonia, etc., there is no necessity for closing the windows. These conditions are also benefited by fresh, cold air.

## CHAPTER XXXIII.

### CLIMATIC TREATMENT.

WE have seen that the vast majority of tuberculous patients are amenable to home treatment; if they are to recover at all, they can accomplish it without leaving their home surroundings. The autopsy findings showing that many persons have healed tuberculous lesions in the lungs and pleura although they have never undergone a course of institutional or climatic treatment, prove clearly that tuberculosis is curable in all climates. But there are undoubtedly indications for certain forms of climatic treatment in tuberculosis, though they are not as imperative, nor as necessary for the average case as the laity and part of the profession believe. In this chapter we shall attempt to review the indications, and point out the limitations of climatic treatment.

Climatic treatment of tuberculosis is probably older than any other method which has survived the recent advent of scientific medicine. The ancient Greek and Roman, as well as the medieval Arabic physicians were great believers in the efficacy of certain climates in the control and treatment of phthisis. The first thought that enters the mind of the average modern physician after diagnosing a case of tuberculosis is "Where should I send the patient?" If the physician is negligent in this regard, the patient will surely ask him "Must I leave the city?"

It is, however, a fact agreed to by all entitled to an opinion that recent studies of the effects of various climates on the incidence and the course of phthisis have not resulted in discovering a region on the habitable globe which can be relied on to cure or improve all incipient or a substantial proportion of advanced cases of the disease. Whenever geographical, topographical, meteorological and clinical data are correlated with demographic data for a given locality, and conclusions drawn that a very high percentage of cases recover when sent there, there are at once shown other facts which prove conclusively that under climatic conditions diametrically opposed to these, the proportion of recoveries is about the same. For these reasons many physicians have gone to the opposite extreme and claim that climate need not at all be considered as a therapeutic agent in the control and cure of phthisis.

**Economic Aspects of Climatic Treatment.**—Other reasons militating against the extensive utilization of certain climates may be mentioned. Bearing in mind that the bulk of consumptives are recruited from the poorer strata of society and that even those who had been self-sup-



porting before they were attacked by the disease often become dependent soon after that event, it is evident that the economic factor is to be given great weight in this connection. Indeed, climatic treatment is as expensive as institutional treatment; it is even more beyond the reach of most patients because modern municipalities provide, as a rule, institutions for the tuberculous, but hardly any supply funds with which patients may go to distant parts of the country and support themselves for a considerable time.

This economic aspect of climatic treatment is too often disregarded by physicians who tell their patients, irrespective of their financial condition, to go to distant regions. Those who cannot raise the funds and must stay at home become despondent and the prognosis is often aggravated as a result of it. Some of them go with meagre funds to Colorado, Arizona, California, etc., and the result is even more disastrous. Very few physicians heed the warnings of experienced men like E. S. Bullock,<sup>1</sup> who says: "I must emphatically maintain that no consumptive should ever be sent away if it is not certain that he will have as good care and management in the distant climate as he could obtain near home."

**Cost of Climatic Treatment.**—Thompson Fraser,<sup>2</sup> who has made a study of this problem in Asheville, N. C., and reported his observations in the Public Health Reports, shows that it must always be borne in mind that there is a clear relation between income and recovery in tuberculosis. When leaving for some climatic region, the patient must be prepared to provide himself with the proper requisites. If he lacks funds he should not undertake a trip which not only exhausts his resources, but does him no good; he should rather stay at home. He points out that at Asheville, and this holds good for nearly every other climatic resort in this country, the expense is about as follows:

The cost of room and board varies within wide limits. From his observations at Asheville, board of fair quality with room costs from \$10 to \$12 a week at the houses which are licensed to take tuberculous patients. The price depends to some extent on the location of the rooms, the more desirable ones costing more, while less desirable rooms may be had for \$8. The "extras," Fraser points out, amount to almost as much as the cost of the room and board, including, as they do, additional food, milk, eggs, reclining chair, physicians' fees, medicines, thermometers, blankets for cold weather, laundry, and everything that comes under the item of "incidentals."

Fraser's conclusions are that the cost to the patient for a period of ten months, or forty-three weeks, at \$8, \$10, \$12 a week would be \$344, \$430, \$516, respectively, not including the extras, just mentioned. A minimum of \$700, therefore, exclusive of carefare, would be a more just estimate of the expense for the rather arbitrary period of ten months. If the patient is accompanied by some member of the

<sup>1</sup> Jour. Amer. Med. Assn., 1909, lii, 1973.

<sup>2</sup> Public Health Reports, September 18, 1914, xxix.

family, it may be decided to keep house instead of to board, but this will not prove more economical in most cases.

The estimate for room, board, and treatment for a period of ten months applies especially to those cases which can be benefited by a comparatively brief stay. If the disease has made greater inroads, and a longer stay is necessary to produce results, the cost of extras and perhaps of nursing may be prohibitive to the average consumptive and it is wiser to remain at home where suitable food, care, and comforts will more than outweigh the benefits of climactic factors if unassisted by these essentials.

Climatic treatment is thus a luxury available for the chosen few, while the vast majority of sufferers from tuberculosis must perforce remain in their homes for treatment.

**Effects of Change of Environment.**—Looking with a sane and unbiased view on the problems of climatic treatment of phthisis, we find that it is undoubtedly an important adjuvant to our efforts at curing our patients. Even physicians who practise in cities and have good results with home treatment are often impressed with the salutary effects of a change of surroundings. One has but to note the effects on a patient who has been kept at home for several months, and all available hygienic, dietetic, and therapeutic measures to control the disease have been taken, yet the patient has been going steadily downward. A change in surroundings is decided upon and he is sent out to the country, preferably a place the patient selects, provided there are no strong objections to it. It makes no difference whether the locality selected is at the sea coast or inland, in a forest or a desert, on a high altitude or the plains; it is immaterial whether the number of sunny days calculated by the weather man or by the owner of the resort in the neighborhood, is small or large, whether it is foggy, or even frequently rainy—the results are often astonishing. After remaining there for a few months, the patient returns greatly improved, in some cases even apparently cured. These are the facts which every observing physician is bound to meet in his daily practice and cannot be controverted by statistics or opinions of famous clinicians. But it is clear that in such cases it is not the meteorological or topographical conditions which are altogether responsible for the good results attained by the change.

Carefully analyzing the results obtained by patients under my observation, I have arrived at the conclusion that the complex phenomena grouped under the title “change of environment,” or the psychic and biological response of the organism to a change in surroundings, play here a greater role than the difference in the composition and density of the air, or the number of sunny and foggy days. The change in environment acts as a new stimulus, reinvigorates and calls forth the dormant vital forces of the patient.

Suggestion is a factor in climatic treatment of tuberculosis which has not been given the credit it deserves. The patient has heard that

a consumptive cannot recover in the city, and, when unable to leave for any reason for some place reputed to be efficacious in this direction, he becomes despondent. Many brood over it to an extent as to negate all other therapeutic measures. Once they are sent away, all potential and inherent vital forces are stimulated; despondency is replaced by a feeling of hopefulness, accompanied by an increase in the appetite, improved assimilation of food, diminution in the cough, etc. This is proven by the following facts which have come under our observation:

Patients leave their homes where they have been under the tender care of relatives and have had good and properly prepared food, to the mountains or the sea coast where they are compelled to live in cheap boarding houses or hotels, in which the food given them is far inferior to that which they had been getting at home. Yet they thrive and gain in weight, while at home they had been wasting progressively. Others go to hotels and boarding houses which, for obvious reasons, allege in their advertisements that the in reality much-coveted consumptives are barred. In fear that when coughing the proprietor of the hostelry is liable to discover their ailment, the patients promptly cease coughing. In many cases the gain is only temporary and after the so-called acclimatization, the "climate wears out." Brown<sup>1</sup> says that it is rarely advisable for a patient to remain in any climate without change for more than eight or nine months. But the gain is immense in a large proportion of cases. The disease often takes a turn to the better, or the patient is carried over an acute exacerbation and given an opportunity to recover his inherent vital forces.

This effect of a change of environment is often seen in patients, themselves natives or residents of agricultural districts; even high mountainous regions, who have become sick with tuberculosis, and coming to the city to consult a physician improve in spite of the fact that climatic conditions are undoubtedly inferior. But there was a change of environment.

That it is not entirely the climate *per se* which is responsible in all cases which improve by a change, is acknowledged by most authorities on medical climatology. Henry Sewall<sup>2</sup> points out an antagonism between the vital effects immediately attendant on a change of climate and those, often totally different in character, which may develop during permanent residence. "In short, a change of scene, irrespective of the character of the environment, has often temporarily a mysterious influence for good on the living organism. The first vital reactions to new climatic conditions involve especially the nervous system, the final effects are dependent on the modified metabolism of the individual organs, and this may or may not be conducive to the efficiency of the body as a whole." Brown puts it pointedly when he says that without doubt many of the effects attributed to climate can be ascribed to change of climate.

<sup>1</sup> Osler's Modern Medicine, i, 488.

<sup>2</sup> Klebs' Tuberculosis, p. 664.

The writer has observed patients who left a favorable climate where they have done badly, for an unfavorable one, where they soon improve wonderfully. Many immigrants who become tuberculous in New York City, try institutional treatment and fail to improve. A longing for their native land overtakes them and they return home where they remain for some months and return to this country cured. We have observed numerous instances of this kind in New York. From personal observations, the writer can testify that the hygienic, sanitary, economic, and social conditions in southern Italy, Hungary, Russia, and Poland, where these patients go, are inferior to those in which they live in New York. Indeed, tuberculosis in those countries is more ravaging than here; is more often fatal. Nor are there sufficient accommodations for dependent consumptives. Still, many immigrant patients, who fail to get relief in the many excellent public sanatoriums in this country, in the mountainous regions of Colorado, Arizona, or the beautiful parts of Southern California, go to some large or small city in southern or eastern Europe and, after remaining there for several months, return apparently cured and able to work.

There is no doubt that in such cases it is not the climatic conditions that helped, but the confidence they placed in their native lands, in the home surroundings, in the caressing tenderness of loving relatives, etc., which was instrumental in awakening the reparative forces of the organism.

There are other reasons for sending patients, who can afford to go, to some region with a favorable climate. It is very often difficult to enjoin complete rest and freedom from the worries and anxieties of every day life in the home of the patient. Nor can he be kept from the temptations of city life. These objects may be accomplished by removing him from his home environment into some secluded country place. The patient is to be told that he will have to remain away from home for several months and he should not leave unless he has sufficient funds for the purpose. His relatives are to be warned against informing the patient of any troubles at home. To this must be added the regular hours for meals, rest, exercise, etc., which are followed implicitly in the country, but often disregarded in the city with its temptations. I have had results which were astonishing with patients sent away in this manner.

With some patients institutional treatment is best for these reasons, as will be shown later on, while with others the reverse is true. In fact, many patients are better off when sent out into the country, than when sent to closed institutions.

**Where to Send Patients.**—Experience has shown that for the vast majority of cases of incipient and uncomplicated phthisis it makes little difference whether they go to a mountainous region or to lowland, to the sea coast or inland, to a moderate or cold region; the effect is practically the same, as long as they are taken away from their homes and placed under favorable surroundings away from the troubles of

home life. There is no climate which cures consumption, the many laudatory advertisements of institutions and railroad companies notwithstanding. The fact that nearly all successful sanatoriums, located as they have been in such a diversity of climatic environments, show practically the same proportion of cured, arrested, improved and last but always least, dead, proves conclusively that if the climatic conditions are a factor, they are of least importance.

A careful perusal of Guy Hinsdale's prize essay on *Atmospheric Air in Relation to Tuberculosis*, which is one of the best books on the subject, and most impartial because the author is not anxious to boost some region or institution, shows clearly that climate is of little therapeutic importance in tuberculosis. He admits that good results are obtained in cloudy regions, as, for instance, in the Adirondacks, and at Rutland, Mass. He has no objection to sunshine because the moral effects of bright sunny days and plenty of them are very great. As to the question of temperature and humidity, Hinsdale concludes that the majority of incipient cases do best in dry and cool places "not warm enough to be relaxing, but not so cold as to be repellant and restrict exercise and out-of-door life." The old ideas about equability of temperature, at least between the temperature of mid-day and midnight, are not of great importance; all mountainous stations show great variations in this respect. Some variability tends to stimulate the vital activities, but in older people and those who are feeble, great variability is a disadvantage. Hinsdale denies that altitude *per se* has any great influence. It is of benefit mainly because it is incidentally associated with mountain life, with more sun, less moisture and scattered population. One statement made by this author should be reprinted with heavy type in all discussions on the subject. "That a place is frequented by consumptives does not prove that it is a desirable place for them."

**Mountain Climates.**—When a change has been decided upon, the first thought which enters the mind of the patient, as well as that of the physician, is whether a high altitude is best. High climates have been popular for centuries; even ancient physicians, who believed that phthisis is invariably fatal, sent their patients to the mountains when feasible. Most of the modern sanatoriums are located in regions of high altitude.

We do not know why high climates are beneficial for consumptives. Various hypotheses have been formulated to explain it, but none have been proven. The purity of the air is beyond question; the absence of massed population assures freedom from air contamination. Humidity is also less frequent, though not as rare as some would lead us to believe and many sanatoriums are located in regions which are notorious in this regard. The air is cool, even during the summer, especially in regions of 4000 feet or more above sea level. But the cold is not felt as acutely even during the winter owing to the greater diathermancy. The ozone, of which many writers of past generations spoke so much,

has been found to be worthless. There is very little ozone, and even if there were more we do not know that it would do much good to the patients.

The diminished atmospheric pressure and rarified air has been considered beneficial by increasing the mobility and expansibility of the thorax. It promotes deeper, fuller, and more frequent respiration. But how much of this is due to the outdoor life, and whether outdoor life at lower altitudes has not a similar effect on consumptives, has never been satisfactorily investigated.

The effects of high altitude on the hematopoietic organs and tissues have been investigated and some have found an increase in the amount of hemoglobin, others, a polycythemia, still others an increase in the number of leukocytes, etc. Webb and Williams<sup>1</sup> have found an increase in the lymphocyte or mononuclear element of the blood as an effect of high altitude. Some authors, notably Bartel, Bergel, Marie, and Fliessinger, have seen in this increased lymphocytosis in tuberculosis a defensive attempt on the part of these blood cells, while others see in it a demonstration that the lymphocytes contain a lipolytic ferment which destroys the waxy coat of the tubercle bacillus. Minnie E. Staines, T. L. James and Carolyn Rosenberg<sup>2</sup> confirmed these findings in Colorado. They found that at an elevation of 6000 feet the larger lymphocytes are absolutely increased in the circulating blood by at least 20 or 30 per cent. in both man and monkeys. Webb, Gilbert and Havens<sup>3</sup> found an increase in the blood platelets in tuberculous human beings and monkeys and that at high altitudes the increase is even more pronounced. But that these blood platelets contain or supply opsonins, or that they play a role in the cure of tuberculosis has not been proved. On the whole, it appears that the hematologic studies of phthisical subjects are contradictory, and it has been shown that the conflicting findings have been due in a great measure to errors in technic. It may be stated that the hypotheses promulgated by some authors have not been confirmed by facts observed by other investigators.

Some have maintained that the proliferation of connective tissue in the lungs, the true reparative process in phthisis, is enhanced by a residence in the mountains. But von Muralt, who formulated this theory, has not given any substantial and convincing proof.

Even the statistics tending to show that deaths due to tuberculosis are less frequent in mountainous than in other climates have not withstood scientific tests. It appears that tuberculosis was rare in the Rockies, the Andes, etc., as long as the population was sparse, the inhabitants leading an outdoor life, etc. But since cities have been established at high altitudes and social conditions favoring the development of phthisis created, the disease is not infrequent among the indigenous population.

<sup>1</sup> Trans. Nat. Assn. Study and Prev. Tuberc., 1909, v, 231.

<sup>2</sup> Arch. Int. Med., 1914, xiv, 376.

<sup>3</sup> Ibid., 1914, xiv, 743.

It is thus clear that economic and social conditions play the same role in the cure of tuberculosis in the mountains as they do in the plains or at the sea coast. On this point all authors are agreed. When a patient goes to a high climate, penniless, and starves there, he will succumb just as quickly as he does in the slums of the city. If he works in Phoenix, Denver, etc., while the disease is active, he may breathe all the rarified air, expand his chest to an extreme degree, and still succumb just as quickly as in the city. It is only those who can afford rest, good nourishment, and careful medical supervision who are benefited by life in a high altitude, and most of these are also doing well in other climates.

**Indications for High Climates.**—High climates are no panacea for tuberculosis; in some cases they are not an unmixed blessing. They have their indications and contra-indications.

Patients in whom a positive diagnosis of active phthisis cannot be made, but who nevertheless show symptoms and signs of the disease—in other words, the so-called “suspects”—may be sent to the mountains for a short or long stay on the principle that they need a rest anyway. But we must be careful and not suggest such a vacation to those with limited means. I have seen self-supporting artisans ruined, their children committed to asylums, while the father was sent away to the mountains without a positive diagnosis of tuberculosis. That they returned within a month or two reinvigorated and in excellent health, was not sufficient to justify the sacrifice; the same result could have been obtained by less costly means. It is different with the well-to-do, who mostly court a vacation.

A large number of neurotics, anemic and debilitated individuals who are in constant fear of tuberculosis, and in whom a diagnosis has been made by some physician, but careful examination fails to elicit any symptoms and signs pointing to a lesion in the lung, are nearly always benefited by a stay in the mountains. Phthisiophobia, which may be considered a distinct syndrome common in modern times, should be treated in the mountains when patients can afford the change. They may remain under the impression that they have been cured of tuberculosis, but this does not make any material difference, as long as they are relieved.

Many of these “suspects” and “phthisiophobiacs” may have been cases of abortive tuberculosis in which the physical signs were indefinite or absent. The rest in the mountains and the change of environment undoubtedly contribute to their recovery.

Incipient cases of tuberculosis with few constitutional symptoms gain considerably by a change for a mountainous climate. The appetite improves, the anemia vanishes and they often gain in weight better than they would have in the city with its temptations. The patients are also freed from the troublesome solicitations of their relatives and friends which are often more a detriment than a help to recovery.

Active phthisis in the moderately advanced stage which does not improve under home treatment for any reason may be sent to the mountains for a prolonged stay. It is at times surprising to see marked improvement manifesting itself soon after their arrival in the country. Fever is no contra-indication, provided it is not of the hectic or terminal variety, or due to some complication which may be aggravated in a high altitude. Occasionally a pleural effusion showing no tendency to absorption will disappear after a stay in the mountains. F. L. Knight preferred patients of phlegmatic temperament to the nervous, with irritable heart, frequent pulse and inability to resist cold.

Of course most tuberculous patients who can afford the expense should be sent to the country, preferably the mountains, during the hot and humid summer months.

**Contra-indications.**—As was already stated, high climates are like a double-edged sword and may be harmful. As a general rule it may be said that hopeless cases, running an acute course with hectic or high continuous fever, with a rapid extension of the process in the lungs, profound emaciation, edema of the extremities, etc., should not be sent, for obvious reasons. It is a great pity to send them travelling great distances, which aggravates their already bad condition, to suffer or die among strangers. Their relatives are also to be considered. Upon hearing of the desperate condition of the patient on his arrival at his destination, they may have to go to see him.

Some of these progressive and apparently hopeless cases take a turn to the better with careful home treatment; the fever abates, the appetite improves, the strength begins to return. At this stage it may be well to send them away to the mountains where the improvement which began in the city is enhanced by the new surroundings. At any rate, they do not lose by the change and, when they can afford it, it may contribute greatly to their ultimate recovery. But they need experienced nurses to take care of them.

Dyspnea is a strong contra-indication to a mountainous climate. It is often not considered and the results are disastrous. Consumptives with dyspnea due to pulmonary emphysema, asthma, and fibroid phthisis, all of which mean cardiac dilatation; or due to cardiac hypertrophy of a high grade, fatty degeneration of the heart muscle, nephritis, arteriosclerosis, etc., should not be sent to a high altitude. F. I. Knight objects to persons over fifty years of age. Tachycardia, when the pulse is much over 100 per minute, and not slowing down after a long rest, is also a strong contra-indication.

Amyloid degeneration of visceral organs, advanced laryngeal, intestinal, and peritoneal tuberculosis are contra-indications. This is not because the climate is harmful, but the hopelessness of the case precludes sending the patient far away from home. Schröder, whose experience has been very large, warns against sending patients with signs of commencing cardiac weakness and with strongly accentuated neuroses to an altitude of over 1000 meters above sea level.



In selecting patients for high altitude, we must not put very much weight on the climatic action on the pulmonary lesion; it is its influence on the heart, bloodvessels, and nervous system that is important. If distinct disturbances in the structure or function of these organs are found, we must warn the patient against high climates. If there are strong reasons for sending him there, it must be done slowly—sending him first to a medium altitude and watching the effect, and when no harm is done he may be permitted to go higher and finally, if he bears it well, he may go up as high as 6000 feet or more above sea level. It is obvious that these experiments can only be made with economically independent patients.

It has been repeatedly stated that hemoptysis is more likely to occur in high altitudes than on the plains, but this is not substantiated by facts observed by physicians with extensive experience in the mountains. All available evidence tends to show that pulmonary hemorrhages are no more frequent on mountains of moderate height (2000 to 5000 feet) than in lower regions. Some authors, like Turban, state that it is even less frequent.

The writer has sent many patients with strong proclivities to bleed while in the city to the mountains, and with the improvement in the general and local conditions, the tendencies to hemoptysis also disappeared. I have often been shocked by the advice given to patients who happen to get a hemorrhage while sojourning in the mountains, to leave at once, and they are in fact taken, while still bleeding, on a long journey. Moribund patients are thus brought to the city occasionally.

Hemoptysis may occur in the mountains as well as in lower regions; it has not been proven that it occurs more frequently in the former places than in the latter. It seems, however, that the results of a copious hemorrhage may be more often serious in the mountains, especially in patients with impaired circulations, as has been shown by F. C. Smith.<sup>1</sup> His statistics show fifty-six deaths from pulmonary hemorrhages out of a total of 524 patients treated at the U. S. Public Health Sanatorium at Fort Stanton, New Mexico, with an altitude of 6231 feet. Ten per cent. of deaths from pulmonary hemorrhages are not seen in other places.

**Sea Climates.**—Ancient physicians recommended sea voyages for consumptives. English medical men of the first half of the nineteenth century considered long sea voyages indicated in many cases of tuberculosis. The fact that they have recently been abandoned shows that they have not met with success. But we often meet with patients who want to take a trip around the world as soon as they are told that they are tuberculous. In other cases in which it is desirable to remove the patient from his home surroundings the most feasible place is at the sea coast. In fact, there are many cases in which, as we have just mentioned, high climates are contra-indicated, and the patient,

<sup>1</sup> National Assn. for Study and Prev. Tubere., 1908, iv, 246.

anxious for some decided change, asks whether a sea coast resort is suitable for him. As was already emphasized, we must always consult the preference of the patient and send him to the place he chooses, unless there are strong reasons against it.

It is obvious that the air on the high seas is pure and free from dust and microorganisms; but near the coast it is greatly influenced by the land climate as well as by the industrial conditions in nearby cities. In fact in some coast cities it is overloaded with dust and soot owing to factories in the neighborhood.

But its moisture serves the purpose of equalizing the temperature; the seasonal differences are less pronounced. However, to this there are many exceptions, and before selecting a sea coast resort, it is best to inquire carefully into the local meteorological conditions.

According to Schröder,<sup>1</sup> sea air has a profound influence on the heart and bloodvessels. The cardiac activity is increased and the pulse slowed. He explains it by the action of the strong air currents and the greater heat conductivity of the moist air; despite the decrease in perspiration, the skin is better cooled and the bloodvessels contract. Reflexly, this causes a greater cardiac activity and the peripheral bloodvessels dilate, causing hyperemia of the skin. The result is strong circulation of the blood from the visceral organs to the periphery. The higher air-pressure causes slower, but deeper respiration, favoring better metabolism and increased excretion of carbon dioxide. The activity of the skin and especially of the mucous membranes is greatly augmented.

Sea voyages are not to be encouraged. "The vicissitudes of sea travel," says Guy Hinsdale, "the narrow cabins and the difficulty of obtaining a suitable diet, even such common requisites as milk and eggs, should be enough to condemn sea voyages. Tuberculous patients ought not to travel more than is absolutely necessary. Imagine the bacteriological condition of a consumptive's stateroom, for instance, at the end of a month's voyage. What sea captain or steward would ever put such a cabin into sanitary condition for the next passenger?" Then it must be borne in mind that sea sickness is liable to do much harm. I have seen many hopeful cases of tuberculosis take a bad turn after a sea voyage during which they suffered from sea sickness.

As a therapeutic measure sea voyages are therefore to be condemned. But patients who are known to bear the travel well, and do not suffer from sea sickness, may be permitted to cross the ocean when necessary. They are, however, to be warned against slow steamers, the sooner they get across the better; and they must be told that it is best for them to spend the greater part of the time on deck, and avoid the close cabin and the stuffy smoking-room.

Empirically, it has been found that incipient cases without pronounced constitutional symptoms often do very well at the sea coast,

<sup>1</sup> Brauer, Schröder, and Blumenfeld's *Handbuch d. Tuberkulose*, ii, 1914, 335.

provided they observe the rules of healthful life. A slight tendency to hemoptysis is no contra-indication, but those who show proclivities to copious hemorrhages, especially in the advanced stages, should avoid the sea coast. Fibroid phthisis, as well as cases of tuberculosis with extensive pulmonary emphysema, are better off at the sea coast than at the mountains and I have seen cases relieved or improved, though in inland climates they had been doing badly. Similarly cases with cardiac and renal complications, which cannot be sent to high altitudes, should be sent to the sea coast when a change is decided upon. Mild implication of the larynx is no contra-indication. The cases of asthma and tuberculosis, in which dilatation of the heart is a strong feature and which are not relieved, or are harmed at a high altitude, should be sent to the seashore where they often recover their strength in a marvellous manner. The same is true of senile consumptives with rigid arteries and rigid chests, in whom paroxysmal attacks of cough and expectoration are occasionally very annoying. They are often benefited by a stay at the sea. Phthisis with chronic bronchitis in which the amount of expectoration is excessive, is relieved at times in a sea climate. Mild forms of neurosis and metabolic disturbances, such as gout, diabetes, obesity, etc., when complicated by tuberculosis, do well at the seashore.

Of course, far advanced cases with hectic or high continuous fever, or with laryngeal, intestinal and renal complications, as well as acute progressive cases, should not be sent to the sea coast but should be kept at home.

**Desert Climates.**—There yet remains to speak of desert climates in which many patients in this country have been cured by "roughing it." These regions may be of low or medium altitude. But their most important characteristic is the capriciousness of meteorological conditions; the changes are quick and extreme. The air is pure—there are usually not enough people to contaminate it—but it is frequently filled with dust and sand, especially after strong winds and storms. Of sunshine there is plenty, often to the detriment of the patient, who finds it hard to contrive a shelter against it.

Because of the frequent changes in the weather, strong, often violent winds, these climates make very great demands upon the reactive powers of the patient, and lead to excessive expenditure of vital force. They are therefore suited only for those endowed with strong constitutions and who have ample recuperative powers. The very young and the very old and those with delicate constitutions, should not be sent to the desert. Moreover, patients of the class just mentioned as proper cases for desert climate, are not satisfied with climate alone. They demand, as a rule, also social life and amusements to distract them, and these they cannot get in those regions.

It has been found empirically that patients with phthisis complicated by bronchitis and pulmonary emphysema, who expectorate excessively, often do well in these regions. Patients with phthisis compli-

cated by renal disease may also do well, provided there is no arterio-sclerosis. Occasionally, we meet a patient in a far advanced stage of the disease who has been "given up," but he decided to discard all comforts and pleasures of life and leaves for some desert region, and within a couple of years returns in excellent condition. These cases are rare, but they do occur. Unfortunately, they admit of no generalization.

**A Warning.**—Before leaving the subject of climatic treatment of phthisis, I want to emphasize the fact that it is not only good air, but also good residence and above all good food that the patient must have if he is to recover. These three in combination are very difficult to obtain. William Garrott Brown, an American historian, who succumbed to phthisis after making a vain fight against the disease, thus describes his experiences:

"It is now seven years and more since I began my quest for a place and an arrangement to breathe freely and constantly the right kind of air, and eat in abundance the right kind of food, yet I can say with perfect honesty that I have not yet found anywhere the combination of these two factors of cure worked out satisfactorily at moderate cost for me and such as I am." He points out that American cookery is peculiarly exasperating—"that is to say, the cooking of such Americans, doubtless the majority, as can be induced to 'take boarders,' and particularly such as can be induced to take boarders who are sick. Many of these last, by the way, are such as have already failed to minister acceptably to boarders who are well. There is, as a rule, not merely unenlightened American cookery, but cookery simulated by no aspiration and but little competition; cookery seasoned with a lax indifference; cookery without any compelling need to be better, and with an obvious reason for being as careless and unlaborious as it can be and continue to be endured. To take 'lungers' at all, it would seem, confers rather than incurs an obligation. For is not that surrendering the chance of any other kind of gainful hospitality?"

These are the reasons why many patients who have done well at home take a turn to the worse after a sojourn in the country for a few months. Physicians should bear this food problem in mind when sending their patients to boarding houses in the country, and when the place selected has an ideal climate but does not have the facilities for proper housing and feeding the patient, he is safer at home under a carefully regulated open-air treatment, as was already described.

## CHAPTER XXXIV.

### INSTITUTIONAL TREATMENT.

**Sanatoriums.**—We have shown that success in the treatment of tuberculosis can only be attained by gaining the confidence and the coöperation of the patient and retaining them over a long period of time, till the termination of the case. The old adage that rest, proper nourishment and fresh air are effective as curative agents, holds good today. But these can only be of benefit when taken methodically and adjusted to the special requirements of each individual case. The tuberculous patient is usually an individual who has not led an exemplary hygienic life, as is proven by the fact that the error of his ways has been instrumental in reducing his natural and inherent resisting forces against the ravages of the tubercle bacilli. He must, therefore, be guided into a healthful mode of life. He must also be cared for in such a manner as to preclude the dissemination of the seeds of the disease among those who come into contact with him.

These are some of the reasons why there have recently been established institutions with a view of solving the complex prophylactic, therapeutic and social problems of tuberculosis. In these "sanatoriums" the patients are under the constant supervision of especially trained physicians who scientifically and methodically guide them along climatic, dietetic and specific lines of treatment. The rules of rational life are minutely enforced and the discipline is of a military character in practically all well-conducted institutions.

As soon as a diagnosis has been made, the problem is at once presented whether the patient should be sent to one of these sanatoriums or may be cared for at home with an equal outlook for ultimate recovery. In deciding this question it is necessary to take into consideration many factors which are but rarely thought of.

**Scope of Sanatoriums.**—Dettweiler established his first sanatorium in Germany in 1859, at a time when tuberculosis was considered incurable because of the teachings of Laennec and the experience of ancient physicians. In this country Trudeau established the first sanatorium at Saranac Lake in 1884 and met with considerable success, discharging cured patients, a thing which was in those days considered impossible. With the evolution of our knowledge of the etiology, pathology and therapy of the disease, the role of the sanatorium has been greatly enhanced. It was expected that it would prove of great prophylactic value by affording places for the segregation and isolation of the sick bacilli "carriers;" that it would prove

of immense therapeutic value because it was assumed that modern methods of climatic, dietetic and specific treatment can only be carried out under the careful supervision of especially trained physicians; that it would prove of great educational value, teaching the patients a healthful mode of life which is in itself an important weapon in the struggle against the disease, and which may be followed by them after their discharge from the institutions.

With these aims in view, numerous institutions have been established in nearly every country of the civilized world at an outlay of immense sums of money for buildings, equipment, and maintenance. In some countries the State or private insurance companies have provided the funds for the sanatoriums. The fact that within recent years the mortality from tuberculosis has decreased was striking proof of the valuable results attained and the sanatoriums were given the lion's share of the credit.

But at present, after these institutions have been in existence for over thirty years, we hear inquiries from many competent sources whether they have done all, or the greater part of what has been expected of them. Articles like that of Edward S. McSweeney,<sup>1</sup> Medical Superintendent of the Sea View Hospital in New York, "Are We Getting Proper Value from Our Plant and Expenditure for the Tuberculous?" are becoming more and more frequent in our medical journals. Considering that immense sums of money have been invested in these institutions, it is but proper to inquire whether they have brought returns along therapeutic and prophylactic lines commensurate with the investment.

**Limitations of the Usefulness of Sanatoriums.**—It seems that the pessimism as to the value of sanatoriums displayed at present is mainly due to the fact that too much was expected from them. They are no panaceas for phthisis. Some enthusiasts who have advocated their erection and raised funds for the purpose have in fact promised too much and when at present these institutions do not come up to the extravagant expectations of some, they are altogether condemned. This is as unjust as the extreme enthusiasm of those who claimed that sanatoriums will solve the tuberculosis problem. In an official report signed by Clifford Allbutt, Lauder Brunton, Arthur Latham, and William Osler,<sup>2</sup> on the value of sanatorium treatment, it is stated: "In many cases, owing to the severity of the disease present, it must be useless; that in a few instances it is actually harmful; and that in many cases this method of treatment need not be carried out in an institution."

Before pointing out the cases in which the sanatoriums may be utilized with benefit in the treatment of phthisis, we shall enumerate some of the shortcomings of this method of treatment:

The number of sanatoriums is inadequate, and we cannot expect

<sup>1</sup> Medical Record, 1915, lxxxvii, 94.

<sup>2</sup> Lancet, 1911, ii, 180.

that there will ever be a sufficient number to provide for all tuberculous patients, just as we cannot expect that all suffering from active disease can be induced to enter and stay within the institutions till the termination of the affliction. In the available institutions there is hardly place for 5 per cent. of the existing proper cases. To provide accommodations for all suitable cases in the United States, several billions would have to be invested in buildings and equipment, and then at least \$100,000,000 annually for maintenance. Even the most enthusiastic of those engaged in the campaign for the control of tuberculosis are not hopeful of ever raising such enormous funds.

Sanatoriums are expensive, and it is problematical whether the results attained within them could not be achieved in the vast majority of cases for a lesser expenditure with home treatment. It costs at least \$1.50 per day to maintain a patient in a sanatorium. The experiment has never been tried on a large scale to spend that much money on a large group of patients treated in their homes consistently for many months.

It appears that only the very rich or the very poor can afford institutional treatment for months under present conditions. The former can pay any price, and the latter are cared for in enlightened cities by the State, municipal or philanthropic institutions. But there is a large middle class which will only reluctantly agree to be treated as public charges, as is the case with clerks, small merchants, professional persons, etc., who have been self-supporting till stricken by the disease. They cannot undertake to pay at least \$20.00 a week for several months and at the same time provide for those dependent on them. Neither are they inclined to enter a State or municipal sanatorium and associate with persons who may be distasteful to them. Only when the disease has advanced far, often beyond repair, and all their own and their friends' resources have been exhausted, do they decide to enter sanatoriums as a last resort, and even then they often leave soon after entering because the surroundings are distasteful to them. This is the main reason why so few incipient cases derived from these classes are entering sanatoriums.

It is very difficult to induce patients in the incipient stage of the disease to enter sanatoriums because they maintain that they feel quite well and resent the idea that they must live among "sick," or among "consumptives," and they often leave soon after entering for these reasons. The strict discipline, especially the unavoidable institutional atmosphere, is distasteful to the average human being who will resist all attempts to place him in an institution as long as he can. The policy of admitting only hopeful cases and discharging bed-ridden or dying patients does not meet with the success worthy of the effort.

Many patients refuse to enter sanatoriums because they do not want to have the stigma of tuberculosis which, they allege, will stick to them throughout their life and may interfere with getting employment under present conditions of private and municipal phthisiophobia.

It can be stated without fear of meeting proofs to the contrary that, on the whole, sanatoriums do not show better lasting results than properly conducted home treatment. In this country, hardly any State or municipal sanatoriums have published satisfactory reports with comparative statistics showing the results attained as compared with a similar group of patients treated in their homes. The most competent compilations of statistics have been published by Lawrason Brown and Pope<sup>1</sup> about the discharged patients from Saranac Lake, and by Herbert Maxon King<sup>2</sup> of the Loomis Sanatorium. To be sure, Brown shows that five, ten, and even eighteen years after discharge some of the patients were found alive and even efficient at their occupations. But the average life of the consumptive outside of the institution, under any mode of treatment, has been found to be between six or seven years. Stadler<sup>3</sup> reports that five years after the onset of the disease one-half of tuberculous patients are found who are able to work without sanatorium treatment. There are similar statistics available for other countries, and I have no doubt that in the United States, we would find conditions the same. King's conclusion as to the value of sanatorium treatment is that his inquiry "clearly demonstrates the uncertainty of apparent immediate results of treatment."

This uncertainty refers mostly to relapses, which are to be expected when we consider the undulating course of phthisis, with its periods of remissions and of acute or subacute exacerbations. The few investigations that have been made of patients discharged from sanatoriums in New York show distinctly that a very high proportion have suffered from relapses of the disease, despite the fact that they have been found "apparently cured," or "improved" at the time of their discharge. Many have to be readmitted because of these relapses, and it has been said that the cure is so good and attractive that many patients like to take it several times.

In estimating the problem whether sanatoriums bring returns commensurate with the money invested in their erection and maintenance, we must deduct those cases which suffer relapses, for obvious reasons. And when we do this in addition to combining with them those who have been discharged because the sanatorium was of no benefit to them, and also those who died, we discover that the cost per successful case is enormous and hardly attractive to municipal and State authorities.

The educational value of the sanatoriums is beyond question, teaching, as they do, objectively the rules of healthful life. But the patients of the lower social strata who make up the bulk of dependent consumptives, cannot, as a rule, continue along the hygienic lines which they have learned. Returning to the tenements, with rooms without windows or baths, coupled with a low earning capacity, one can not

<sup>1</sup> American Medicine, 1904, viii, 879; Ztschr. f. Tuberkulose, 1908, xii, 206.

<sup>2</sup> National Assn. for Study and Prev. Tuberc., 1912, viii, 82.

<sup>3</sup> Deut. Arch. f. klin. Med., 1902, lxxv, 412.



live in the manner he learned in an institution. Relapses, which are likely under all circumstances, are inevitable for these reasons alone.

On the other hand, the recent educational campaign carried on by the various antituberculosis agencies has done all that can be done along educational lines. In fact, the dispensaries with their social services, the day and night camps, etc., achieve educational as well as therapeutic results which are, from a certain viewpoint, superior to and more extensive than those of the sanatoriums, and at less cost.

Let us not overestimate the prophylactic value of the sanatoriums. It was hoped that by segregating consumptives, sources of infection would be isolated. But we have already shown that this was a vain hope. Only "incipient" cases are admitted—as far as they can be found and induced to enter in time—while advanced cases, which are the most dangerous because they expectorate miriads of tubercle bacilli, are rejected. The statement that the institutional treatment is the predominant cause of the decline in the death rates from phthisis, which has been expounded by Newsholme<sup>1</sup> with such vigor, is not supported by facts. Newsholme's figures have been demolished by Karl Pearson,<sup>2</sup> one of the most competent authorities to judge statistics. In Germany—the home of the sanatorium—this claim has been abandoned during recent years. As was pointed out by Cornet and Robert Koch at the Antituberculosis Congress in London, there were at least 226,000 persons disseminating tubercle bacilli in Germany, and only 20,000 were cared for in institutions, and of these latter only 4000 expectorated bacilli. This number could not have had any perceptible influence on the morbidity and mortality from tuberculosis. In the United States conditions are the same.

From the clinical standpoint, we are not in possession of reliable statistics showing that the mortality of patients who have been treated in sanatoriums is lower than that of those who have been cared for in their homes. We have already mentioned that the institutions in the United States have not published comprehensive data along these lines, excepting those by Lawrason Brown and King. In Germany, although long and apparently learned books and articles have been produced, they are just as much in the dark about this problem as we are in this country. The reasons are that the material is not comparable. A drastic illustration may be cited. In the selection of cases it is aimed at admitting only those in the incipient stage. The result is that at Grabowsee 45.2 per cent., and at Melsungen 97 per cent. of the patients have not shown any tubercle bacilli in the sputum. Ulrici reports that in 40 per cent. of the patients at Mulrose he could not make a positive diagnosis of tuberculosis, and Leube says that many patients who are admitted to sanatoriums in Germany

<sup>1</sup> The Prevention of Tuberculosis, London, 1908.

<sup>2</sup> The Fight against Tuberculosis and the Death Rate from Phthisis, London, 1911; Tuberculosis, Heredity and Environment, London, 1912.

are, when examined by military surgeons, found fit for the army, and accepted.

It is obvious that statistics of such "consumptives" will show good and lasting results of treatment. In their book on the prognosis of tuberculosis Kuthy and Wolff-Eisner, reviewing the subject, say that exact and scientific data are not available to prove the value of sanatorium treatment; and Newsholme, who is a great believer in the benefits of institutional treatment, also says that there are no exact and comparable data available to prove it.

**Causes of Failure of Institutional Treatment from the Therapeutic Viewpoint.**—While institutional treatment undoubtedly has its advantages, which will be shown later on, it is by no means the best and clinicians cannot approve of all the methods pursued in sanatoriums. The fact is, wholesale treatment of such a complex disease as phthisis is not ideal. Individualization is here of greater importance than in most other diseases. Says Albert Robin:<sup>1</sup> "One of the disadvantages of the sanatorium is that it applies too often arbitrary principles to patients whose disease can only be relieved by individualized methods. It is for this reason that the practitioner who knows how to adapt the treatment to each of the small number of patients under his care, and to take cognizance of the temperamental indications, is qualified to manage a case of tuberculosis as well as, if not better than, the sanatorium doctor who has under his care a large number of patients of whose individual idiosyncracies he is ignorant, at least for a time, and must therefore have a strong tendency to subject them all to the same method of treatment." Charles L. Minor says: "By seeing our patients too frequently—as we do if we live in the same house with them—we are apt to lose that objectivity in our attitude toward them which is so important; and to lessen rather than increase our control." This refers to private sanatoriums, in which the patients must be catered to, if they are to be retained for months. In state and municipal sanatoriums, where the poor and dependent patient faces starvation if he leaves the institution, the trouble is of a diametrically opposite character. The fact that a large proportion of patients leave before the physicians discharge them shows that they cannot be satisfied.

This lack of individualization in treatment is seen in many ways in the sanatoriums which are hotbeds of therapeutic hobbies. But this is usually not as harmful as the uniformity of the diet in institutions. Mass feeding is difficult at best and can only be carried out in jails, where the inmates have no choice, or in armies during war. To subject to the same dietary tuberculous patients in different stages of the disease with different individual capacities for digestion and assimilation, who have been brought up on and adapted to different kinds and preparations of foods, is bound to meet with failure. For this

<sup>1</sup> *Traitement de la tuberculose*, Paris, 1912, p. 67.

reason we find that complaints about the quantity and quality of the food are universal in public sanatoriums, and to some extent in private institutions where food is served *a la carte*.

It can hardly be expected that municipal, State, and philanthropic sanatoriums should supply food *a la carte*; it will always be *table d'hôte*. And for this reason resentment on the part of the patients is to be expected. To be sure, these institutions are always filled and there is a long waiting list. But when patients leave before they are discharged, we may safely assume that the cost incurred during several weeks or months for their maintenance was to a large extent wasted.

In American municipal sanatoriums of the large industrial cities the failure in this regard is even greater than in other countries, because we must care for tuberculous immigrants of various nationalities whose tastes differ extremely as regards food and its preparation, as is shown elsewhere in this book.

These are some of the drawbacks of sanatorium treatment. It is for these reasons that the municipal and State sanatoriums in many cities of the United States are not filled with a desirable element, but contain a large proportion of underserving individuals. "My efforts are not going to be devoted to coddling tramps and other parasites," exclaims in despair Dr. Edward S. McSweeney, the Medical Superintendent of the Sea View Hospital in New York. These are also the reasons why the best elements of the tuberculous population in this country will always have to be cared for in their homes, as is the case at present.

**Indications for Institutional Treatment.**—But there are many cases of tuberculosis which cannot be treated in any other place but in institutions. In fact any one with experience in a large city is convinced that tuberculosis cannot be managed without the aid of institutional treatment. Of the cases which are suitable for sanatorium treatment and would be lost without it, we may mention the following:

Among well-to-do patients we meet with many who, for various reasons, cannot be cared for in their homes. To send them to the country without control may prove disastrous, because the foolish and reckless rich show at times greater lack of self-restraint than the stupid poor. They are best cared for in private sanatoriums in which most of the drawbacks of the public institutions are eliminated. They may be sent to sanatoriums for a short stay, over the hot summer months, or for outdoor treatment for the relief of an acute exacerbation, etc.; or for a long period till the disease is arrested. Great care should be taken that they do not become egocentric, excessively introspective, or hypochondriacs, which is not unusual.

Among the poor and those who have become dependent because of the disease, we meet with a large number of patients who have no family to care for them during their illness and, with or without funds, they are unable to find lodgings under present conditions of rampant

phthiophobia. Many boarding houses bar persons who cough; and at times even near relatives are overtaken with a sense of stupid fear of infection and want to get rid of the unfortunate patient. For these there is left nothing but to go to a well-regulated sanatorium.

There is a large number of phthical patients who notoriously lack will power to carry out the most important of the measures prescribed for them and, remaining in the city, they are apt to be tempted by the opportunities for gay life or even excesses. They are better off in sanatoriums.

On the other hand, there are many who show all willingness to do everything that is conducive to the cure of the disease, but they have not the funds to pay for capacious rooms in a desirable part of the city, for good nourishment and medical attendance. Tuberculosis is after all the most expensive of diseases, not only for the special and costly nourishment and residence which are required, but mainly for the long time the patient must remain idle and the savings of years may be exhausted before he can resume work. While most of these can be and are well cared for in the clinics, the day and night camps found in every large city at present, we meet with many who, for obvious reasons, are better off in sanatoriums, at least for short stays.

Most phthical patients should leave the city during the hot summer months, and those who cannot raise the funds for the purpose are proper charges of the sanatoriums. Indeed, if the sanatoriums were not filled with lazy, undeserving tramps and vagrants who remain for years in the institutions, and when discharged from one, soon gain admission to another, they could well care for the just mentioned class of patients. It seems to me that the German system of admitting tuberculous patients for three or four months is much superior to ours, where they are often kept indefinitely. The result is that the patients must wait for months before beds are vacant for them and truly incipient cases, left without proper care while waiting for admission, become advanced.

The longer we are up against the problems presented by tuberculosis in the city, the more we are convinced that the public sanatoriums ought to be converted into hospitals which admit patients on short notice, keep them for a few weeks, a month or two, until they regain their strength and are fit for treatment in the clinics. Patients who suffer from acute exacerbations during the long, chronic course of phthisis could then be cared for. Inasmuch as municipal institutions are now in abundance near cities, this could easily be accomplished.

But sanatoriums still work on the theory that they are to cure their patients, which they cannot do in more than 5 or 10 per cent. of cases, which is, in fact, not more than home treatment accomplishes.

## CHAPTER XXXV.

### DIETETIC TREATMENT.

**Economic Aspects of Dietetics for Consumptives.**—Because phthisis is accompanied by wasting of the body it requires careful, generous, and at times excessive nourishment with a view to covering the deficit created by the extravagant drain resulting from the toxemia, fever, loss of appetite, disturbed digestion, faulty metabolism and concomitant emaciation. Cornet suggests that the rapid waste of the tissues tends to hasten absorption of the proteins surrounding the tuberculous foci and thus at the same time inhibits the natural process of healing by means of induration and also furthers the peripheral dissemination of the bacilli. Inasmuch as the disease finds most of its victims among the poor and destitute, or causes destitution and despondency in those who have been self-supporting before its onset, the dietetic problems are not only of a physiological nature but also have important economic bearings. It is self-evident that a dependent consumptive must not be prescribed food which is beyond his reach financially.

In my experience the dietetics of phthisis are, in fact, more dependent on the financial resources of the patient than on the careful calculation of the number of calories contained in the various foodstuffs. Considering the variety of dietaries which have been urged by various authors in this disease, and that each author claims good results with his method, it is obvious that no specific diet has been devised which will suit every case. In fact, all that can be stated is that tuberculous patients need food, just like other persons who are underfed, but they usually need more of it.

**Need for Individualization of Diet.**—Most of the studies in the dietetics of phthisis have been carried out in sanatoriums, some of which have had sufficient funds for an extravagant diet, while others with meager finances have shown similar results. But the lessons from institutional experience are not applicable in their entirety to patients treated in their homes. On the other hand, the time-honored advice given to tuberculous patients: "Eat plenty of milk, eggs, and meat," is often decidedly harmful to those who follow it implicitly.

There is great urgency for individualization of the diet in phthisis; it is important that the diet should be adapted to the needs of the patient and not to the disease. The "personal equation" counts for more than the disease.

There is no doubt that the failure of institutional treatment of

phthisis is, in a large measure, due to negligence in this regard. Wholesale feeding is usually disastrous for human beings. The food in first class *table d'hôte* restaurants is usually unbearable to the average person when relied on continually for a considerable time. It is impossible to make up a menu which will suit the palate, digestive capacities, and functions of one hundred patients in an institution where they must remain for months. The difficulties are greater with tuberculous patients whose gastric functions are very often deranged. Tuberculous patients cannot be treated like soldiers in the army or prisoners, if we are to succeed in our aims.

It is not true that two kinds of food of different composition, but theoretically of the same nutritive value, will invariably be of the same digestibility or produce the same effects. It may be calculated in the laboratory that a portion of beef steak, roast beef, poultry, sausages, stew, cheese, potatoes, cereals, bread, milk, eggs, etc., contains a certain proportion of proteins, fat, and carbohydrates and will liberate a certain number of calories when burned in the body. In fact, we know that the intrinsic value of three eggs is equivalent to about 100 grams of red meat, while 100 grams of bread is approximately equal to one egg, or 30 grams of beef, or 200 grams of potatoes or 280 grams of milk. But very often a consumptive assimilates three eggs more easily than 100 grams of beef, or 300 grams of bread. At times the patient assimilates 250 grams of milk better than 200 grams of potatoes. Because of the personal equation many patients refuse to thrive on scientifically prepared dietaries. An Irishman resents spaghetti, an Italian refuses Irish stew, a German prefers sausages to the English roast beef, etc.

For these reasons, in prescribing a diet for a patient we must always take into careful consideration his habits of life, the foods upon which he has been raised and his personal likes and dislikes. Even when a change is imperative, it is dangerous to institute it suddenly, and we must make a strong effort to fit the diet to the one the patient has been used to. The factors which should guide us are the presence or absence of anorexia, fever, constipation, diarrhea, etc.

**Superalimentation and Forced Feeding.**—With a view of replenishing the wasted tissues, especially in those who are by nature bad eaters, it has been suggested that superalimentation or even forced feeding is indicated in most cases of phthisis. It has been observed that occasionally an emaciated patient gains in weight under such a regime, and some authors have advised that all sufferers from phthisis should be “stuffed.” Even Debove’s method of introducing food through the stomach tube into those who would otherwise not consume large quantities of nourishment, was in vogue for some time till it was found that the gain in weight which forced feeding produced in some cases was not necessarily an indication that the lesion in the lung had improved. It was also found that many patients under forced feeding, with or without the stomach tube, may gain in weight and improve otherwise

for some time when suddenly the gastro-intestinal tract rebels, and within a few days they lose more than they gained in several months.

**Estimation of the Nutrition of the Patient.**—In our attempts at estimating the results of certain dietetic methods in tuberculosis we cannot always be guided by the scientific determination of the number of calories ingested by the patient every day; nor even by the quantity of proteins, fat, and carbohydrates which the patient has consumed. Attempts along these lines have proved futile in practice; they have not given us a diet which will suit all, or the vast majority of cases. It seems that only clinical observation of the individual patient, his state of nutrition, his digestive capacity and the assimilability of the ingested food are of value in this regard.

We aim at increasing the amount of nourishment so that the patient shall gain in weight, and remain stationary at somewhat above his usual, or normal weight before the onset of the disease. While in the vast majority of cases a gain in weight is a good index of the value of the diet, it is, however, often liable to mislead. We very often see patients who prove that fattening by no means goes hand in hand with enhancing the resistance against the tuberculous toxemia. We also meet with cases with hardly any gain in weight, in fact remaining under the standard weight, yet the lesion in the lung heals and recovery is good.

“The main object of dietetic treatment,” says Brown,<sup>1</sup> “is to enable the patient to regain his lost weight, but not to make him a flabby, breathless mass of inert fat.” Excessive nourishment, which increases the weight of a patient more than two or three pounds per month on the average, is apt to result in an overload of fat and water without any utility. We should strengthen but not fatten the patient. “When a workman has to perform hard work he eats meat,” says Daremberg,<sup>2</sup> “The consumptive has to perform a very hard task, the task of repairing his wasted body.” In fibroid phthisis obesity is not rare—“obésité toxique” of the French—and is often more annoying to the patient than the symptoms in the respiratory organs.

In the average case we may judge the progress of the disease by following the weight of the patient, provided we also take other factors into consideration. With the increase in weight there should also be an increase in strength; physical examination should also show regression of the signs in the lungs, the cough should be ameliorated, and the quantity of sputum decreased. With such signs, a slow and persistent gain, finally reaching ten to fifteen pounds higher than the patient's normal weight before he was attacked by phthisis, indicates that we may be satisfied that the diet is good.

**Do All Tuberculous Patients Need Special Diets?**—A large proportion of phthisical patients, probably one-third of all, have good appetites and digestion. In fact, even febrile consumptives are seen without

<sup>1</sup> Osler's Modern Medicine, i, 482.

<sup>2</sup> Les différentes formes cliniques de la tuberculose pulmonaire, Paris, 1905, p. 149.

anorexia which accompanies nearly all other fevers. The prognosis is good as long as they retain their gastro-intestinal functions. They may be told that a moderate increase in the quantity of food they have been accustomed to eat is sufficient and, when possible, they should increase somewhat the quantity of proteins and fats, provided the stomach does not rebel.

If the constitutional symptoms are in abeyance or disappearing and the signs in the lung show that the lesion is cicatrizing, we should not worry about a lack of gain in weight, or even when they show a few pounds less than their normal weight. A patient with a good appetite and digestion needs no special diet; he should eat just like any other person, or a little more, if he can without inconvenience. On this point all authorities agree today. Thus, King<sup>1</sup> says: "In the absence of certain complications, a diet which would suffice for the same individual under normal conditions of life will doubtless, with very slight modifications, meet the requirements in the presence of tuberculosis, the more especially during that period of the disease when constitutional symptoms are either absent or but slightly manifest." Paterson,<sup>2</sup> whose patients work at graduated labor, gives them "a liberal diet which consists of the ordinary food which the working classes provide for themselves when they are in a position to afford it." In fact, patients who tend to become excessively fat have their diet reduced in quantity.

On the other hand, patients who lose progressively in weight and strength, are anemic and debilitated despite the rest which is rigidly enforced, need more and better food if they are to recover or hold their own in the struggle with the disease. But even here superalimentation must be carefully adapted to the digestive capacity of the patient.

It may be stated as a general rule that the suggestion of some authors that in such cases the patients must consume between 4500 and 6000 calories daily is a dangerous one. Experience has taught that one who will not recover or hold his own on a diet of 3500 calories, will not recover at all. Professor Fisher<sup>3</sup> says: "We may feel satisfied that given proper food elements, the average tuberculous patient can be successfully nourished on 3000 calories per day. In other words on no more than is usually consumed by the sedentary man." N. D. Bardswell and John E. Chapman<sup>4</sup> have arrived at the same conclusion after a thorough experimental study of the subject.

**Variety.**—The first principle to be observed in the diet of the tuberculous patient who is losing weight is variety, both as regards nutritive principles as well as appetizing qualities. There is nothing more abhorrent to a tuberculous patient, and to a large extent to all sufferers from chronic diseases, than homogeneity of diet. No limited and exclusive diet can keep a patient well for any length of time because

<sup>1</sup> Sixth Intern. Congr. Tuberc., 1908, i, 719.

<sup>2</sup> *Ibid.*, p. 893.

<sup>3</sup> *Ibid.*, p. 694.

<sup>4</sup> Diets in Tuberculosis, London, 1908.



it does not respond to the urgent demands of the different organs and tissues of the body. It does not stimulate the secretions of all the digestive glands. If an exclusively animal diet is taken, only the gastric juice is stimulated, while the saliva, pancreatic juice, bile, and intestinal juices are not utilized and, remaining free in the gastrointestinal tract, are apt to act as irritants and produce diarrhea which is exhausting, or constipation which is harmful in other ways.

We often meet with patients who have been given diet lists in which four or five meals are listed for the day. But any appetite they may have had before the list was consulted promptly disappears, because it shows the foods which have been given them for months without any appreciable variation. Many patients who have followed the injunction "plenty of milk and eggs" have engendered such an aversion to these articles that the mere mention of an egg is sufficient to disturb the slight appetite for other foods which was called forth by hunger. It is always advisable to consult the patient as to the kind of food he prefers or longs for and, if there are no contra-indications, to give it to him.

**Precautions to be Taken when Overfeeding Patients.**—Before a patient is urged on to a course of superalimentation certain precautions are to be taken: He must be carefully examined with a view of ascertaining whether or not he can stand additional feeding. Those showing signs of arteriosclerosis, nephritis, gall-stones, nephrolithiasis, or gout should not be allowed superfeeding, especially with animal proteins. It is likely to throw a considerable strain on the kidneys or even produce albuminuria. The condition of the stomach is to be ascertained, and those having dilated organs, or disturbances in the tonicity and motility of the viscus are to be treated for these troubles when practicable. The appetite is of great importance. Although we may succeed with some patients in urging them to eat irrespective of the appetite, we will fail with many.

Proper preparation of food goes a long way in counteracting anorexia; Dettweiler said that the kitchen was his pharmacy. It is better to give the patient small quantities of each of several dishes, well and appetizingly prepared, than large quantities of one or two dishes. The fact that the food value is theoretically sufficient in the latter case does not alter matters. With some patients animal food should predominate, with others eggs and with still others, milk. The diet must be frequently changed, especially when the digestive tract shows signs of rebellion.

With well-to-do patients these are simple matters, but with the poor the problem is often hard to solve. The writer usually sends for the mother, wife, or sister of the patient and gives her directions along these lines.

Bearing in mind that the disease is likely to last for months, if not for years, we must spare the digestive organs, the cornerstone of phthisiotherapy, as they have been called, and not overburden them

with work. The first imperative principle is proper mastication. But regularity in meals is of the same importance. The menus of some authors mention six and more meals a day, which are excessive in my experience. Three, at most four meals a day are sufficient for most patients, and afford some rest to the stomach between the meals. At all events, the stomach must be given a complete rest during the night, which can be done by avoiding all food between 9 P.M. and 7 A.M.

**Proteid Foods.**—Experimental researches of Richet and Héricourt and others have proven conclusively that when ingested raw, animal foods have an especially beneficial effect in tuberculosis. The specific effect seems to reside more in the juices of the meats than in the fiber. Herbivorous animals, like the cow, are more prone to tuberculosis than carnivorous animals, as the dog.

The best source of proteins for a tuberculous patient is animal food, the proteins of vegetable origin are not as easily assimilated. Meats possess all the qualities which are necessary for the nutrition of the consumptive. To be sure there are some who maintain with Kellogg<sup>1</sup> that a low protein diet is productive of better results, and urge vegetable proteins in the dietetic management of the malady. It is, however, an every day observation that the animal proteins do not tax the digestive organs to excess and, excepting in those who suffer from some form of dyspepsia, they can be taken by most consumptives without difficulty in comparatively large quantities. Beef, mutton, lamb, poultry, game, fish, oysters, eggs, milk, cheese, etc., offer a wide range of choice for variety.

Those who have no natural abhorrence for raw meat may have it with great benefit—zomotherapy was at one time very popular, and should be utilized, when tolerated. Some patients are not averse to taking small pieces of raw beef, dipping it in tomato sauce and eating it. It is, however, better to mince or chop it, and eat it between two slices of bread as a sandwich, but it should be seasoned to taste. The vast majority of patients, however, prefer roasted or boiled beef, mutton, poultry, etc. It must be mentioned that when roasted or broiled, meats should be rather underdone and, on the whole, they should be changed often.

But it should never be excessive; we cannot rely on animal foods exclusively in nourishing a tuberculous patient. To supply a patient with 5000 calories per day, it would be necessary to gorge him with six and a half pounds of meat, or thirty-six eggs, or five quarts of milk, or two pounds of cheese. This would be too much—no human being could take it with impunity for any length of time. For this reason other foodstuffs are necessary in addition to the animal food. The most the average consumptive should have is about three-fourths to one pound of meat, and when taken raw, it should not exceed one-

<sup>1</sup> Sixth Intern. Congr. Tuberc., 1908, iii, 740.

half-pound per day. When this is taken with one pound of bread, three eggs, one quart of milk, eight ounces of potatoes, and four ounces of fresh vegetables, the diet is complete.

A consumptive needs more protein foods than a healthy person because the disease destroys the tissues, especially the muscles, and there are no better tissue builders than proteins. But we must not give them at the expense of other foods. It is unnecessary, even dangerous, to give more proteins than is required for repairing the tissues; otherwise it is likely to prove more disastrous than to a healthy individual. These evils are, as the researches of Chittenden, Mendel, Folin, Herter, Metchnikoff, Tissier, Combe, Kellogg, Turk, and others show: (1) that protein which is not used for tissue building is not "burned clean," as are fat and carbohydrates, which yield merely water and carbon dioxide, but leave behind "clinkers" in solid form—for instance, uric acid; (2) that meat proteids also contain such "clinkers" in their extractives, which are superadded to the similar products from the metabolism of proteins in the body; (3) that all protein which is not absorbed is subject to putrefaction in the intestinal canal, and gives rise to toxins which are partially absorbed, and produce injuries of various kinds to the organism (Irving Fisher).

**Milk.**—Milk has been considered for centuries a good food for consumptives—Aretæus already spoke of it in this connection. It contains more than 10 per cent. of nutritive matter, albumin, fat, sugar, and salts. But this does not mean that it is good to use it exclusively for our patients as has been done in the well-known "milk cures." If we wanted to supply all the requirements of a patient it would be necessary to make him ingest five to seven quarts of milk per day. In a few weeks his stomach would be dilated two or three times its normal dimensions.

But with other foodstuffs it is excellent because its nutritive principles are easily digestible in the stomach and intestines, and it contains no toxic substances. It is just as good for a patient with fever as for one who is afebrile.

It is best given between meals in the form of drink and may be added to many other foods, especially cereals. But it must not be abused; patients who gorge themselves excessively with milk lose their appetite for other foods. Between a pint and a quart of milk per day is to be considered the maximum for the average patient.

There are patients who do not bear milk very well. In some it provokes lactic and butyric acid fermentation in the stomach; this viscus becomes dilated and the complicating hyperchlorhydria favors spasmodic contraction of the pylorus. In others, the milk clots excessively in the stomach, large solid curds are formed which irritate the mucous membrane and cause nausea and vomiting. In some patients the milk passes the stomach without difficulty, but it produces trouble in the intestines—gaseous distention and diarrhea. I have seen many cases of diarrhea in consumptives, which were thought

to have been caused by intestinal ulcerations, but which disappeared with the withdrawal of milk from the diet.

The milk may be rendered more digestible by diluting it with alkaline waters, or lime water, but then the total quantity consumed must be reduced. It is usually more easily digested when given with some cereal, like oatmeal or rice. Atwater found that milk is more easily digested when it is part of a mixed diet. When consumed alone the proportion digested was: proteins, 91.2 per cent.; carbohydrates, 86.3 per cent.; and fat, 92.8 per cent. When milk and bread made up the diet, the amount digested was: proteins, 97.1 per cent.; carbohydrates, 98.7 per cent.; and fat, 95 per cent.

Fermented milk is often more easily borne in large quantities when the pure article is not sustained. We may try koumiss, keffir, or the various preparations of buttermilk which are at present supplied by most milk dealers at reasonable prices, or may be prepared at home with cultures or tablets of lactic acid bacilli.

Cheese is an excellent food for consumptives. But we should avoid the highly seasoned varieties. Cream cheese and ordinary pot cheese contain considerable nutritive elements and do not provoke cough or gastric irritation.

**Eggs.**—Eggs are considered an excellent food for tuberculous patients by the profession and the laity. In assimilability they exceed any known food excepting milk and oysters. It has been found that raw eggs make no demand on the stomach and may pass through, hardly altered, into the duodenum. They contain enormous quantities of albumen and fat. The white of an egg consists of pure protein which is as digestible and nourishing as that of beef; the yolk contains 25 per cent. of fat, 15 per cent. of protein, and also nuclein, lecithin, iron, and salts. Eating one dozen eggs per day, a consumptive could feed himself, and pushing it to twenty eggs he would absorb the equivalent of two and a half pounds of beef, because an egg of 50 grams is equivalent to about 35 grams of moderately fat beef, or 128 grams of cow's milk. In other words, they contain over 700 calories per pound; the whites yield 250 and the yolks 1700 calories per pound. But an exclusive egg diet is just as bad as an exclusive meat diet. Too much fat is introduced into the stomach and congestion of the liver is the result, while with an exclusive meat diet, congestion of the kidneys occurs.

In my experience it makes little difference in what form eggs are given to consumptives with good digestive functions, but I discourage them from ingesting a half to one dozen raw eggs a day as some are apt to do. The mode of preparation has no influence on the digestibility of eggs, excepting that when hard boiled they remain somewhat longer in the stomach.

Those who suffer from derangement of the function of the stomach and the liver do not bear eggs very well and they may have to be discarded. The same is true of patients who have an idiosyncrasy

to eggs and get colicky pains in the abdomen, vomiting or diarrhea from an egg.

Four to six eggs per day is about the maximum which a patient should be allowed, if we are to retain the functions of the stomach and liver. In most cases less should be given.

**Fats.**—While the amount of fat necessary for the average consumptive has been exaggerated by many authors, it is nevertheless a fact that a diet containing a surplus of easily assimilated fat is the best. It must, however, be borne in mind that the capacity for digesting and assimilating fat varies with the individual. In some patients an increase in the amount of fat is immediately followed by gastro-intestinal disturbances. Many people cannot digest fat meats like bacon, ham, etc. We have already mentioned that many patients have shown intolerance for fat even before the onset of the disease.

I have found that butter is superior for our purposes and it has given me results as good as cod-liver oil which has been popular for centuries. I direct my patients to cut their bread in thin slices and cover them with heavy layers of butter; mixing butter with mashed potatoes and other foods. As much as six to eight ounces of butter can thus be consumed daily by the average patient without gastric or intestinal disturbances. Those who like, and can consume, large quantities of unskimmed milk may get the greater part of their fat in this manner, while cream and certain kinds of cheese are also rich in fat. In looking for sources of easily digestible fat we must not forget fish: Salmon, pompano, sardines, shad, fish roe, caviar, etc., are very good for this purpose. Those who have great tolerance for fat may also take in addition to butter, cream, cream cheese, fat meat, and bacon.

The quantity of fat a patient should consume varies according to the season, the kind of food he has been accustomed to eat, his tolerance of fat and the condition of his gastro-intestinal tract. Of course, those who are obese, and they are not rare among quiescent cases, should be discouraged from eating an excessively fat diet.

It has been my experience that a patient without preëxisting gastric disease can consume six ounces of fat every day for months with benefit. But now and then one is met who shows a decided inclination to fat intolerance. It is my impression that in most cases it is due to the excessive amounts of improper fats which have been forced upon them. It has been suggested by Tibbles that when a patient cannot take fat, the proteins can be increased; 100 grams of proteins will yield 40 grams of fat. Proteins alone will never fatten a patient; 6.5 pounds of lean meat, or 5.5 pounds of lean and fat meat would be required to supply the daily requirements of carbon for an ordinary person; therefore some other source for carbon must be found.

We must guard against quick fattening, "stuffing," of tuberculous patients. Often consumptives are urged to eat plenty and some ingest enormous quantities of food and gain remarkably well. Taking their weight weekly, and finding that it keeps on increasing, they are

encouraged to continue in this manner and at the end of three or four months the gain may be as much as thirty or even forty pounds. But to their dismay they have not been rehabilitated in other respects; they are as yet unable to work and are in fact weaker than before. The weight they have put on is only an added burden, which is not only useless but incapacitating. In addition, they suffer from annoying dyspnea. Physical examination shows that the process in the lungs has not improved; perhaps it has distinctly extended. Carefully and guardedly reducing these patients has often been of great benefit.

**Carbohydrates.**—In the eagerness to supply the body of the patient with proteins and fat, carbohydrates must not be neglected from the diet. They are, as a rule, easily digested and assimilated, and they spare the proteins, thus maintaining the nitrogenous balance or equilibrium with smaller quantities of albuminoids. The best sources of carbohydrates are potatoes, cereals—like oatmeal, rice, etc., which may be taken with milk or cream—pastries, and above all, bread. Cane sugar and maple sugar, which enter into various culinary preparations, are of great value. Daremberg,<sup>1</sup> however, objects to excessive consumption of sweets by consumptives because they are usually dyspeptics who do not stand it very well. He says that those who can take an excessive quantity of sugar may become fat rapidly; but this fattening is not lasting, just as the fattening obtained from an excessive milk diet. The best fattening is obtained from a mixed diet. However, there is no reason against eating sweet desserts, or even candies in moderate quantities, provided they are taken after meals.

**Salts.**—Mineral salts must not be neglected. Even if the theory of demineralization is not well founded, there is no question that the loss of mineral salts is higher among consumptives than in healthy individuals. Iron, lime, soda, magnesia, and the phosphates are best supplied by such foods as bread, flour, oatmeal, rice, sago, tapioca, fresh vegetables, and fruits. All these may be given plain, or, better still, in various other culinary preparations.

**Condiments.**—For their local appetizing effects, condiments, acting as they do as great salivary and gastric stimulants, may be taken, especially by those who suffer from anorexia. Some condiments, like mustard and garlic, contain allyl which assists in the digestion of fats, and is said to be bactericidal in the intestinal tract. At one time garlic was considered a good remedy against tuberculosis. Its active principle, allyl, was even administered subcutaneously.

**Dangers of Overfeeding.**—While the majority of patients stand a moderate increase in the quantity of food fairly well, there are many who are decidedly harmed by it. This is especially seen in those who have been unreasonably induced to increase the quantity of protein foods, such as eggs, meat, etc., thus imposing an excessive and

<sup>1</sup> Loc. cit, p. 157.

often dangerous burden upon the liver, kidneys, etc. In some cases we find that these organs have been decidedly crippled by such a diet.

The symptoms produced by excessive protein consumption are unmistakable: The patient is drowsy for an hour or two after meals, has headache and is irritable. At night he is restless and sleepless, or his sleep is disturbed by frightful dreams. The abdomen is distended, the liver enlarged, and may be tender on palpation, and he has heartburn. Anorexia, bilious vomiting and diarrhea are often seen. Cardiac palpitation and night sweats are at times due to the indigestion thus induced. Because of the plethoric condition, the patients often have epistaxis and also hemorrhoids which contribute to their misery. The urine contains albumin, biliary pigments, indican, and glycosuria is not rare. Arthralgic pains in the joints are often the result of superalimentation. Older clinicians, believing that there exists an antagonism between the gouty and phthisical diatheses, urged excessive nitrogenous diet combined with wines, with a view of inducing sclerotic changes in the diseased lungs. On a similar principle, the excessive consumption of alcohol was advised in former days. The acneiform eruptions on the skin of some tuberculous patients are very frequently due to the excessive protein foods which they consume.

When overfeeding a patient we must watch out for the following danger signals: Failure of appetite and symptoms of flatulent dyspepsia; dyspnea on exertion which is obviously not due to the tuberculous toxemia or the lung lesion; diarrhea, and at times vomiting. If these symptoms are not heeded and forced feeding is continued, irreparable damage may be done, the sheet-anchor of the patient, his power to digest food, is damaged, and his chances of recovery are materially lessened. But this should not deter us from trying to feed the tuberculous patient generously. "Excessive feeding is clearly a vastly better method of treatment than underfeeding, for it at least ensures the consumptive taking enough to repair his waste and to restore his normal power of resistance and recuperation," say Bardswell and Chapman,<sup>1</sup> "The point to realize is, that it is quite an unnecessary hardship for patients to be overfed, and that it may do positive harm."

When these harmful results of unwise feeding are borne in mind, unfortunate patients will not be forced to ingest large quantities of food which may be excessive and dangerous to healthy persons. Especially careful must we be with plethoric, obese, and sedentary consumptives. A dilated stomach which does not empty itself with ease and promptness is particularly to be spared. The dangers of excessive fat consumption have already been dwelt upon.

**Dietaries.**—From what has been said it is obvious that it is not necessary to give detailed dietaries for consumptives. When we aim

<sup>1</sup> Diets in Tuberculosis, London, 1908, p. 49.

at variety as the first requirement for a good diet, it would be necessary to give at least thirty menus to suit the average case. We will, therefore, merely mention some of the foods which may be utilized in attempts at feeding phthisical patients properly. It will be noted that they may eat nearly everything a healthy person can, as long as their malady is not complicated by conditions which alter matters.

**Breakfast.**—Milk, coffee, chocolate, cocoa, or tea. Bread, butter, cream, eggs, bacon, ham, ox tongue, fish (fresh or canned), fruits of any kind. Plenty of butter. Cereals of any kind.

**Lunch.**—Fish, or entrée; meats (roasts, chops, steaks, etc.), poultry, vegetables, custards, puddings, cheese, milk, coffee, fruit.

**Dinner.**—Soups, meats, puddings, poultry, game, fish, all vegetables, puddings, pastries, etc., cheese, ice cream, coffee, milk or chocolate.

Without going into details of the various dishes that may be prepared by a good cook who knows the likes and dislikes of the patient, it can be stated that there is no dish which is contra-indicated in uncomplicated phthisis. A good cook can do more for the patient than all the dietaries which may be printed in a book.

Between the main meals there may be allowed a light luncheon between the early breakfast and the lunch, consisting of a glass of milk and some biscuit. Some are allowed an egg or two at that time, made in some form of punch, taken raw, or in any style, provided it is well borne. Similarly, at about 4 P.M. tea, coffee, or milk may be allowed with some biscuit, etc. At night before retiring, a cup of milk with some crackers is beneficial for some patients. It will be noted that in this manner the patient may have his milk—about one-half to one quart per day—mainly outside of his mealtime, as drinks.

It must be emphasized again that these foods should be palatably prepared and rendered digestible by proper cooking. Otherwise trouble may arise. The quantity to be ingested depends on the personal equation of the patient, although in some cases matters may be forced for some time when indicated, but this should only be done bearing in mind the contra-indications which have already been discussed.



## CHAPTER XXXVI.

### MEDICINAL TREATMENT.

**Importance of Medicinal Treatment.**—The disrepute of medicinal substances in phthisis during recent years is due to several causes. The first and most important is that we have no specific botanical, chemical or physical agent which, when administered to a consumptive, will exert a selective action on the tubercle bacilli, as mercury and salvarsan do on the spirocheta of syphilis and quinine on the malarial parasite. Nor have we a therapeutic agent which will enhance the resistance of the tissues against the ravages of the tubercle bacilli, or neutralize their poisons, or stimulate sclerosis of the affected area. But here we are in about the same position as when dealing with anemia, typhoid, pneumonia, rheumatism, etc. When we find that the salicylates relieve the more painful symptoms of rheumatism, and that iron increases the hemoglobin content of the erythrocytes in chlorosis, that digitalis increases the force of the cardiac muscle, we use these drugs although we know that digitalis does not regenerate destroyed heart valves, and salicylates do not remove the essential cause of acute articular rheumatism. Similarly if we find that creosote, arsenic, ichthyol, etc., have a beneficial influence on some of the annoying clinical phenomena of phthisis, though they do not cure the disease, we must not discard them merely because they do not remove the cause of tuberculosis, or kill the bacilli within the body, or neutralize the tuberculous poisons, etc.

There is another aspect to be considered in this connection. Excepting the chosen few, who have sufficient means to pay for first-class sanatorium treatment and inclination to remain in the institution for months and perhaps years, the bulk of the patients must be treated in their homes. Even if they get a few months of sanatorium treatment in a public institution, they must be treated in dispensaries or by their family physicians before admission and after discharge. The patient is a human being; and when we consider the human element we find that, as a rule, he has no confidence in a physician who has no remedy for his ailment. The dictum "plenty of fresh air, milk, and eggs," he believes he knows as well as the physician. If his medical advisor will not prescribe for him, he will seek remedies from another who is more obliging in this respect, or from an advertising quack. This is not only true of the ignorant, but also, almost to the same extent, of the supposedly intelligent patient.

It cannot be denied that in many respects medicaments, properly administered, act by psychic suggestion. But so do the minute and

detailed directions given, often in writing, about diet, rest, exercise, sleep, etc., in institutions. "Medicinal agents," says G. Küss,<sup>1</sup> one of the most ardent advocates of tuberculin treatment in France, "no matter in what they consist, always inspire confidence in the physician; without them he is helpless. Moreover, by giving the patient in addition to other treatment, a prescription calling for some medicine, we may succeed better in our attempts at keeping him away from the alluring advertisements of charlatans who very often impose on him."

**Harmless Medication.**—The reasons why medicinal agents have fallen into disrepute in medical literature—by no means in the practice of the vast majority of physicians—are manifold. But the most important is perhaps the fact that drugs have been abused. "I regard medication as indispensable in the treatment of tuberculosis," says Rénon.<sup>2</sup> "It has an undoubted good effect on the disease in general and an enormous psychic effect. But there is one important condition which must be realized above all when giving drugs to consumptives—they *must be harmless.*" He illustrates this point by the following instance: Some years ago the acetate of thallium was suggested as an excellent remedy against the nightsweats of phthisis and a trial showed that it did control this symptom very well indeed. But it also had another effect: It caused the hair to fall out and the nails to shed. The patients stopped sweating, but incidentally lost their hair and nails, which was a good reason for resentment. That certain drugs used in phthisiotherapy may have disastrous effects in addition to their influence on the disease or some of its symptoms, must always be borne in mind. In fact it has been stated with considerable truth that 50 per cent. of the dyspepsia in phthisical patients is due to improper medication.

**"False Specifics."**—It is absurd to banish drugs from the armamentarium of the physician because they are "false specifics." As if true specifics are plentiful in other diseases. It is curious that those who label creosote, arsenic, and the iodides as false specifics, and urge specific treatment in the form of tuberculin, are in one breath stating that a specific remedy is yet to be found. "The wanton theory that you can treat with medicines and cure a pneumonia and typhoid fever," says Abraham Jacobi,<sup>3</sup> "but not a case of tuberculosis, has taken possession of the oracular mind of the Colorado-ridden exile doctor. He should know better and do better. There is a drug treatment for tuberculosis, as for other diseases, and he should be glad to avail himself of it. There is no panacea, however, for tuberculosis, as there is none for pneumonia, or typhoid fever. But there are indications, and improvements of condition, and prolongation of life and recoveries."

<sup>1</sup> Gilbert and Carnot's *Therapeutique*, xxi, 594.

<sup>2</sup> *Le traitement pratique de la tuberculose*, Paris, 1908, p. 110.

<sup>3</sup> *American Medicine*, 1905, x, 1063.

**Creosote.**—There are very few sufferers from tuberculosis who have not been given creosote at some period of their illness. Its history is similar to that of tuberculin. Introduced by Reichenbach, in 1830, it was given in very large doses resulting in considerable harm to the patients. It was discarded for this reason, to be reintroduced some twenty-five years ago, and ever since it has held its place in the armamentarium of the physician in general and special practice. Its most ardent advocates do not consider it a specific, but then those urging tuberculin are still looking for a specific for tuberculosis. In the hands of those who have administered it intelligently it has proved the best medicinal agent to relieve some of the most baneful symptoms of the disease.

When administered in the proper cases and in proper dosage, it improves the appetite, stimulates digestion and assimilation, improves nutrition, diminishes expectoration, removing at times its purulent character and disagreeable taste and odor, all of which is sufficient of an encouragement to the average sufferer from phthisis to bestow confidence in the physician and to look forward to an ultimate recovery. This beneficial action of creosote is ascribed by some authors to its power to inhibit the growth, or destroy tubercle bacilli in the gastrointestinal tract, which are inevitably swallowed by every consumptive. It is one of the best gastric and intestinal antiseptics we have. It has been found that part of the ingested drug is excreted by the bronchial mucous membrane and, while it cannot be expected to destroy the bacilli in the lungs—hardly any drug could reach the avascular tubercle, even if it could be given in sufficiently large doses—it exerts there a beneficial influence as is evidenced by the decrease in the amount of sputum brought out, and the diminution in the intensity of the associated bronchitis, laryngitis, and tracheitis.

It is a peculiar fact, not generally appreciated, that creosote often provokes general and local reactions which are analogous to those provoked by tuberculin. Usually with excessive doses, but occasionally also with minimal doses, after taking creosote for several days the patient is overtaken by a feeling of chilliness and fever, pain in limbs, back, and joints, weakness, fatigue, and insomnia. Malaise, gastric disturbances and even vomiting, in patients whose stomach has heretofore not given any trouble, now make their appearance. The part of the creosote eliminated through the bronchial mucous membrane often excites a focal reaction which at times reminds one of the focal reaction of tuberculin. Of course in the case of tuberculin a single dose is often enough to produce this reaction, while in the case of creosote it is only the more or less prolonged administration that is apt to produce this effect. In such cases sanguineous expectoration and even hemorrhage is not uncommon, while the lesion in the lung may be aggravated or even spread. Rales, which were previously absent or scanty, now make their appearance and the general aspect of the patient is aggravated.

If the administration of creosote is persisted in after these symptoms, as I have seen many times, the condition of the patient may be aggravated to an extent as to render the prognosis hopeless in a case that previously had a fair outlook. Smoky urine, like that of phenol poisoning is now seen; the patient complains of a taste of creosote in his mouth. This may be followed by vertigo, profuse perspiration, chilly sensations, and even cyanosis and collapse, as I have seen in one case which was greatly relieved by the discontinuance of the drug.

**Contra-indications.**—Bearing all this in mind we can say that creosote is contra-indicated in all cases in which it provokes gastric disturbances. If after taking moderate doses of the drug the appetite does not improve, it should be discontinued. It is also contra-indicated in all febrile cases in which the temperature is 100° F. or more, and also in all progressive cases, because they are the ones in which general and local reactions are apt to be provoked and spread the lesion in the lungs.

Patients subject to hemoptysis must not be given any creosote; even blood-streaked sputum should serve as a warning for the immediate discontinuance of the drug. Moreover, one must not wait for the appearance of smoky urine, but carefully watch for albumin which is often brought about by creosote. In general, albuminuria is a strong contra-indication to the administration of creosote.

**Indications.**—In all incipient cases in which the appetite is poor and digestion defective, creosote may be given. With the improvement in the nutrition of the patient, owing to cessation of gastric and intestinal fermentation, the local condition in the lungs also shows improvement. In chronic, sluggish, afebrile cases of tuberculosis, especially those characterized by profuse expectoration, creosote is often of immense benefit, if rationally administered. In addition to its good effects on the gastro-intestinal functions, it also diminishes the amount of expectoration, ameliorates the cough, etc., and with the gain in weight and comfort, it has an excellent effect on the psychic state of the patient who becomes more encouraged and hopeful. In fibroid phthisis, characterized by profuse expectoration of purulent material, provided there is no concomitant emphysema, creosote is the best remedy we have. I have seen drying up of cavities, at least temporarily, in some measure due to the proper administration of creosote.

**Administration.**—A good product must be used. Soon after its introduction creosote fell into disuse mainly because of the bad quality of the product. Good creosote, fit for therapeutic administration, must be obtained from the fractional distillation of beech-wood tar. The product dispensed in many pharmacies in this country is obtained from the distillation of bituminous coal and contains many impurities which are not well tolerated. A good preparation of creosote contains 25 per cent. of guaiacol, but many of the products dispensed under this name, even when obtained from beech-wood, contain much less.

It is best administered in capsules which does away with the disagreeable odor. Moreover, the mucous membrane of the stomach and intestines is not as easily injured by creosote as that of the mouth and pharynx, so that the disagreeable local effects are done away with through capsules. Some mix it with balsam of tolu, and it is best given after meals. Those who cannot swallow capsules may take it in this form:

R̄—Creosoti . . . . .	ggt. xxx	2.0
Vini pepsini . . . . .	℥iv	120.0
M. S.—Teaspoonful in water three times a day after meals, gradually increasing.		
R̄—Creosoti,		
Picis liquidæ radicis . . . . .	āā gr. xxiv	1.5
Alcoholis absol. . . . .	℥iij	12.0
Balsam. peruv. . . . .	℥iv	15.0
Tinct. Helianthi annui . . . . .	℥v	20.0
Olei terebinth. rectificati,		
Myrtholi . . . . .	āā ℥ij	7.5
M. S.—Three times a day, one teaspoonful in milk or water one hour after meals.		
R̄—Tannini . . . . .	℥v	20.0
Calcii phosphorici . . . . .	℥v	20.0
Creosoti . . . . .	℥iiss	10.0
M.—Div. in part 40; ft. capsul.		
S.—One capsule three times a day after meals.		

Beverley Robinson has had good results with the following:

R̄—Creosoti . . . . .	ggt. vj	0.5
Glycerini . . . . .	℥j	25.0
Spiritus frumenti . . . . .	ad ℥iij	100.0
M. S.—Teaspoonful in water three times a day after meals.		

This dose may be increased to two or three teaspoonfuls, or, if it is desired to increase the creosote, the amount of it may be doubled. If the whisky is deemed inadvisable, elixir calisaya or the compound tincture of cardamom may be substituted.

Many have administered creosote by inhalation and have obtained good results. In this country, Beverley Robinson introduced this method. He recommends equal parts of creosote and alcohol or, when there is much irritative cough, equal parts of creosote, alcohol, and spirits of chloroform, on the sponge of a perforated zinc inhaler. The inhaler should be used frequently, at first for a few minutes, later gradually increasing the time till it is used from half an hour to an hour at a time, and finally it may be used almost continually during the day and frequently all night. "These inhalations modify sputum favorably, diminish its quantity, lessen cough, thus promoting rest, sleep, and nutrition and general improvement physically, and in some instances appear to be the means through which the patient has gotten rid of tubercle bacilli permanently."

The following are good formulæ for inhalation:

R—Creosoti . . . . .	gtt. vij	0.5
Tincturæ benzoini comp. . . . .	ʒiij	100.0
M. S.—To inhale a teaspoonful from boiling water, three or four times a day; shake.		

R—Creosoti . . . . .	gtt. vij	0.5
Olei pini silvestris . . . . .	ʒiiss	10.0
Olei terebinthinæ . . . . .	ʒiss	5.0
Tincturæ benzoini Comp. . . . .	ʒiv	100.0
M. S.—Shake. To inhale a teaspoonful, from boiling water three or four times a day.		

**Derivatives of Creosote.**—Because of its caustic taste and disagreeable odor creosote is not well tolerated by many patients; even when given in capsules the odor is often penetrating. Guaiacol, the main active principle of creosote, can be given instead, but it is insoluble in water, has an objectionable odor and taste and is a gastric irritant. There have been brought out a large number of preparations which retain most, or all, of the useful qualities of creosote without its drawbacks. These derivatives of creosote are mostly used at present with the same result as with the original drug.

Of these creosote carbonate (creosotal) is perhaps the best. When ingested it breaks up slowly in the intestine, liberating creosote. It may be given in capsules of 5 to 10 drops three or four times a day. Many pharmaceutical houses market globules which are very elegant. It may also be given to patients to be taken in a certain number of drops in water, milk, or coffee; or the following prescription is useful:

R—Creosoti carbonatis . . . . .	ʒiv	120.0
Aetheris . . . . .	ʒiiss	5.0
Alcoholis sol. . . . .	ʒvj	25.0
Vanilin . . . . .	gtt. vij	0.5

M. S.—Fifteen drops in water or in milk three times a day after meals; increased if well tolerated.

In many cases between 30 and 60 grains of creosote carbonate may be given per day.

Guaiacol carbonate (duotal) is another preparation which is very extensively used. It may be given in powder or capsule from 10 to 40 grains a day, or combined with arsenic.

Both of the two above preparations are now sold quite reasonably. But for those who can afford to pay, we have a wider range of choice. Styraol (guaiacol cinnamate) contains a high percentage of guaiacol. Thiocol (potassium-guaiacol-sulphonate) may be given in 5 to 15 grains three times a day in powder, tablet, or capsule. It is a non-toxic, tasteless, odorless powder, soluble in water. Many patients who do not tolerate guaiacol take this preparation very well, and in those who suffer from diarrhea it is to be preferred. But it contains less guaiacol than most other preparations of this class and its action is not as intense as that of the others. In fact, it is sometimes not

decomposed in the intestines, and may be excreted unchanged. For those who prefer their medicine in liquid form and for children, it may be given in the form of sirolin, a 10 per cent. solution of thiocol in orange syrup, which may be given one to three teaspoonfuls three times a day. There is no doubt that many who cannot tolerate creosote or guaiacol take this less toxic preparation very well.

Sir R. Douglas Powell recommends the following:

R—Guaiacol carbonatis, guaiacol benzoatis vel styracol . . . . .	℥iiss	6.0
Calcii hypophosphatis . . . . .	℥ss	2.0
Pulvis tragacanthæ co. . . . .	℥j	4.0
Misce bene, adde guttatim:		
Syr. pruni virginianæ vel elixir aurantii . . . . .	℥ss	16.0
Syr. calcii lactophosphatis vel syr. hypophosphitum co. . . . .	℥j	32.0
Aquæ chloroformi . . . . . ad	℥vj	190.0
S.—One teaspoonful in water or liquid malt three times a day soon after meals.		

R—Creosoti carbonatis . . . . .	℥iv	16.0
Tinet. gentianæ co. . . . .	℥iv	16.0
Syr. pruni virginianæ . . . . .	℥iiij	90.0

S.—One teaspoonful in a wineglass of water or malt extract after meals three times a day. Increase the dose by five drops each second day up to two teaspoonfuls by measure.

**Ichthyol.**—Ammonium sulphoichthyolate or ichthyol has been found very useful in many cases of phthisis. Some authors state that it has a favorable influence on the metabolism, prevents albuminous decomposition and favors assimilation of food. Helmers found that about one-third of the sulphur ingested with ichthyol circulates in the juices of the body; others asserted that it even had a bactericidal action, without hurting the body cells, etc. It may, however, be stated that we do not know the exact pharmacology of this preparation, but that empirically it has been found useful in many cases of phthisis.

It may be given in water 2 to 5 drops three times a day, beginning with the smaller dose and gradually increasing according to tolerance. Because of its disagreeable odor and taste, the drops should be diluted in large quantities of water or milk and given before meals. It may also be administered in black coffee. Or the following formulæ may be used:

R—Ichthyolis . . . . .	℥vj	25.0
Aquæ distil. . . . .	℥ij	60.0
Alcoholis rectific. . . . .	℥ij	60.0
Syr. citr.,		
Syr. aurant cort . . . . .	℥ā ℥iiss	50.0
M. S.—Teaspoonful in water three times a day before meals.		

De Renzi says that the above formula conceals the taste and odor of ichthyol. The following is also of use:

R—Ichthyolis . . . . .	℥iiss	10.0
Syrup. simpl. . . . .	℥v	20.0
Aquæ menth. piper. . . . .	℥iiij	80.0
M. S.—Teaspoonful in a glass of water three times a day.		

In many cases ichthyol improves the appetite, diminishes the frequency of the cough and the expectoration, changing the latter so that its purulent character vanishes. The general condition of the patient improves with the improvement in the nutrition. In some patients the remedy disagrees, causing flatulence, abdominal pains, diarrhea, loss of appetite, and eructation of gases. In fact, as has been shown by Barnes in patients in whom the administration of ichthyol does not immediately improve the appetite, it is not advisable to continue the drug. I can add that diarrhea also shows that the drug disagrees. My patients do not, as a rule, mind the disagreeable odor and taste when given well diluted with water, milk, or coffee.

Ichthyol should be tried in every case of phthisis because it has not the dangerous characters of creosote and arsenic and their derivatives; in fact it is well tolerated in most cases; only gastro-intestinal disturbances occasionally preventing its use.

**Arsenic.**—Arsenic has been found an excellent stimulant of nutrition, a hematinic, reconstructive, and alterative in chronic wasting diseases including phthisis. The various organic arsenic compounds recently introduced were stated to lack the greater part of the toxicity of arsenic while retaining its curative, reconstructive, and antiseptic properties. The advocates of arsenic medication in tuberculosis claim that it increases the appetite; improves assimilation of food, and stimulates the blood-forming organs in addition to its stimulating effects on the nervous system. In short, arsenic is supposed to fortify the tissues against the ravages of the tubercle bacilli.

From an extensive use of arsenic in phthisis the author has not found that it exerts any direct influence on the tuberculous lesion in the lungs, even when administered to patients who tolerate it. The quantity and quality of the expectoration is, however, very favorably influenced; purulent sputum often becoming mucous and greatly reduced in quantity. With the improvement in the appetite and nutrition a great deal is gained—the patient is encouraged. The fever is, however, not influenced, nor are the night sweats. In fact it should not be given to febrile patients.

It may be given as an adjuvant to creosote treatment in the form of trioxide, as in the following formula:

R—Guaiacolis carbonatis . . . . .	ʒv	20.0
Arsenici trioxidi . . . . .	gr. iss	0.1
Strychninæ sulphatis . . . . .	gr. j	0.06

M. ft. pilullæ no. lx div.

S.—One pill three times a day after meals.

It may be given in the form of Fowler's solution, beginning with 2 or 3 drops after meals and increasing daily until 10 drops are taken three times a day.

During recent years various organic compounds of arsenic have been used in phthisis, administered either by mouth or hypodermically. Of these the cacodylates of sodium, strychnin, iron, and guaiacol may



be mentioned. Many of these, as well as atoxyl, are at present sold by pharmaceutical houses in ampoules ready for hypodermic and intravenous administration. But in my experience none of these preparations has any advantages over the inorganic arsenic; the trioxide and Fowler's solution answer all requirements. In fact some of them, notably atoxyl, are dangerous because they are liable to cause amblyopia.

When administering arsenic to phthisical patients certain precautions are to be taken. It should not be continued, especially in large doses, for more than a week or ten days. Symptoms of intolerance may make their appearance, such as loss of appetite, thirst and dryness in the mouth, colicky pains, and diarrhea. In some cases the fever rises as a result of large or even small doses of arsenic. Tachycardia, cardiac palpitation, and insomnia are occasionally observed. It should not be given to febrile patients, and to those showing a tendency to hemoptysis. In fact, if during the administration of arsenic there appears streaky sputum, it should be considered a danger signal and the arsenic is to be discontinued at once.

**Iodin.**—For generations iodine has been used in the treatment of scrofulous children with good results. It has also been found useful in assisting the resolution of pleural adhesions and in the relief of the symptoms of chronic bronchitis, pulmonary emphysema, and asthma. That the iodides have an effect on tuberculous lesions in the lungs is evidenced by the fact that small doses of the iodide of potassium may cause, in persons with incipient tuberculosis, reactions similar to those produced by tuberculin, as was shown by Rondot. In fact many authors recommend it for diagnostic purposes, at least to provoke expectoration which may be examined for tubercle bacilli. Sorel<sup>1</sup> found that tuberculous animals, when given large doses of potassium iodide succumb to generalized miliary tuberculosis, and usually much earlier than the controls.

Some French authors recommend the iodides in most cases of pulmonary tuberculosis, but it seems to be a dangerous drug for the reasons just stated. But in some cases of incipient phthisis without fever the iodides do good, especially in those in whom the tuberculous process has been implanted on emphysematous lungs. This is also true of asthma and tuberculosis—the iodides often control or relieve the nocturnal attacks of dyspnea. But one must always guard against giving this drug to sufferers from the congestive, inflammatory, progressive lesions, and those subject to hemoptysis.

It is best given in a saturated solution of iodide of potassium of which each drop represents 1 grain of the drug. Small doses are to be given at first, 2 to 5 grains, three to five times a day. If no intolerance is shown it may be increased. I have often used some of the organic compounds of iodine—sajodin, etc.—with good results.

<sup>1</sup> Ann. de l'Institut. Pasteur, 1909, xxiii, 533.

**Succinimide of Mercury.**—Mercury has been used in the treatment of tuberculosis for many years. But more recently Dr. B. L. Wright developed a new method of administering it and reported a larger number of recoveries than has been claimed with any other medication. He used the succinimide of mercury hypodermically, in doses of  $\frac{1}{5}$  of a grain given on alternate days increasing the dose guardedly till the limits of toleration are reached. As soon as symptoms of mercurialization appear, or there is a rise in the temperature, anorexia, loss of weight, etc., the dose is either reduced or the treatment is discontinued for a time. In most cases about thirty injections are given, followed by a rest of two weeks, during which period iodide of potassium may be administered. A second series of injections is given to those who tolerate the drug.

I have tried this treatment and found it of immense value in phthisis complicating syphilis, otherwise it is decidedly harmful. As was already stated it appears that when tuberculosis is implanted in a syphilitic subject, the disease is apt to run a very sluggish, chronic course. Fibrosis is very active. In these cases both the iodides and mercury, if intelligently and guardedly administered, may be very efficacious. The succinimide of mercury may be used instead of other forms of the drug. But the doses given by Wright are decidedly excessive—the same results may be obtained by the hypodermic administration of  $\frac{1}{8}$  or  $\frac{1}{12}$  of a grain twice weekly. On the other hand, salvarsan now offers a better means of combating active syphilis combined with tuberculosis than the succinimide of mercury.

**Hypophosphites and Glycerophosphates.**—It will be noted that most of the medicinal preparations mentioned above have their indications and contra-indications, and some are not without danger when improperly administered. The safest medication in phthisis appears to be the time-honored administration of the hypophosphites. Recently the glycerophosphates of lime, iron, magnesium, etc., have been used very extensively on the theory that phthisis is a manifestation of lime starvation and that recalcification and remineralization of the body are of great importance in our efforts at combating the effects of the tuberculous process. There is no doubt that in many cases of phthisis these medicinal substances have an excellent influence on the nutrition of the patient and they are also of use in relieving the anemia which is such a frequent accompaniment of the disease. We may give the official compound syrup of hypophosphites in doses of one to two teaspoonfuls three times a day after meals. The glycerophosphites may be given in any form. Pharmaceutical houses have many elegant and palatable preparations of glycerophosphites in tablet, capsule, and liquid forms which may be used. Their tonic effects are beyond question.

**Cod-liver Oil.**—Physicians of past generations bestowed great confidence in the therapeutic virtues of cod-liver oil in tuberculosis, and many modern practitioners still consider it an excellent thera-

peutic agent. Some have ascribed the curative action of this oil to certain of its constituents. Thus, some believe that it is the iodine which is effective, others see in the bromine the active principle. But careful chemical analysis has shown that there are only traces of these elements in cod-liver oil. The biliary salts, the hepatic ferments, the lipoids, the lecithin, etc., have been stated to be of more value than the fat of cod-liver oil. John W. Wells<sup>1</sup> and others believe that, in addition to the ready absorption of the fat of cod-liver oil, it possesses powers of increasing the absorption of other fats of the food to a marked degree.

The recent intensive studies of the internal secretions have also thrown some light on the action of cod-liver oil in phthisis according to some authors. Thus, Williams<sup>2</sup> recently stated that the superiority of this oil to others is mainly due to the internal secretion of the liver of the fish, which "when introduced into the human economy, acts as a stimulant to one of the normal internal secretory glands, and the secretion of the one so stimulated is inimical to the development of the tubercle bacilli." He believes that only the crude oil contains these active principles and is therefore more efficacious than the refined oil. Iscovesco,<sup>3</sup> from his experimental researches, is convinced that the efficaciousness of cod-liver oil is due to the lecithides which it contains. He treated a large series of animals for four months. Those who got cod-liver oil increased in weight to the extent of 55 per cent; those who got cod-liver oil from which the lecithides had been removed gained only 27 per cent.; those who were given olive oil gained 33 per cent.; others were given oil to which was added 0.5 *pro mille* of the lecithides extracted from cod-liver oil and they gained 56 per cent. The control animals gained only 29 per cent. Williams and Forsyth<sup>4</sup> claim that the unsaturated fatty acids of cod-liver oil tend to disintegrate the waxy envelope of the tubercle bacilli, thus destroying them.

These theories are interesting, and deserve further study, but there is no doubt that cod-liver oil is an important remedy in tuberculosis, even if only for the fact that it contains a considerable proportion of easily assimilable fat, and it may be used as a food rather than as a drug. Patients who do not take animal fats like butter, etc., are distinctly benefited by cod-liver oil.

Cod-liver oil should be given in large doses; to some patients as much as 2 ounces per day may be given and some French authors, like Jaccound, Grancher, and Daremberg, have given more than 4 ounces per day. Some apparently have a marked tolerance for this preparation, and they may utilize it instead of superalimentation. On the other hand, there are patients who cannot tolerate it, and even small doses

<sup>1</sup> British Med. Jour., 1902, ii, 1222.

<sup>2</sup> Practitioner, 1911, lxxxviii, 605.

<sup>3</sup> Compt. Rend. Soc. Biol., 1914, lxxvi, 34.

<sup>4</sup> Brit. Med. Jour., 1909, ii, 1120.

cause eructations, nausea, and oily taste in the mouth. Diarrhea is another of the untoward effects in some who do not bear the oil very well.

**Indications.**—Cod-liver oil is indicated in all afebrile cases of phthisis. All patients who willingly take it, and digest it well in large doses, should be given this oil, without incidentally curtailing their usual amount of other nourishment. It may be continued for a long period of time; as long as the patient is apparently benefited by it and his digestive functions remain normal, the appetite is good and, above all, there is no diarrhea. Patients with fever do not tolerate it as well as those who have no pyrexia. Children with tendencies to scrofula, with enlarged tuberculous glands, especially tracheobronchial adenopathy, and who are as a result underfed and anemic, often derive great benefit from cod-liver oil. It appears that children take it with greater ease, and more often with distinct benefit, than adults.

**Contra-indications.**—Cod-liver oil is contra-indicated in cases in which the patients do not tolerate it in even small doses. The best criteria are the state of the appetite and digestion. As soon as these are deranged, it should be discontinued.

**Administration.**—As long as we consider cod-liver oil merely a fat food, and disregard its other constituents, it is best to administer it in as palatable a form as possible. In former times the crude oil, a product of decomposition of the livers of the cod, was used. Some modern authors even now insist that this form is most beneficial for phthisical patients. But it has a very disagreeable odor and taste and it requires courage on the part of the patient to swallow it. It is also apt to cause indigestion, eructations, diarrhea, etc. The light, or amber-colored oil, prepared by melting fresh livers by a steam process, is less disagreeable and more easily tolerated. It should at first be given in small doses of the Norwegian, light-colored oil, and in case the gastro-intestinal tract tolerates it, the dose is to be increased so that within a few weeks the patient takes four to six tablespoonfuls a day after meals. It should not be forced on patients; when they refuse to take it, or it causes nausea, eructations, diarrhea, etc., it should be discontinued.

It is best that the pure oil should be given and many patients take it easily. With some the odor and taste have to be masked, and this may be done in the following manner: It may be given in orange juice, or in some volatile oil. Many patients take it with ease in coffee or milk. A pinch of salt placed in the mouth before taking it may disguise the taste. Those who are allowed to take alcohol may take some whisky or brandy into the mouth where it is kept for a few seconds without swallowing, and then the oil is taken. Some use peppermint-water or tomato ketchup for the purpose, or orange- or lemon-juice. The difficulties owing to the odor and taste are overcome soon in most patients, and they take it freely.

The various emulsions offer no advantage over the pure oil. If

they contain the indicated percentage of the oil, they are as disagreeable as the pure article, and one who can take an emulsion, can take and digest the oil. The various preparations and "extracts" which are alleged to have all the therapeutic qualities of cod-liver oil without any of its disadvantages, have been found worthless, lacking as they do the fatty substances which are of value for the nutrition of the patient. On the other hand, many of the preparations of cod-liver oil and malt, hypophosphites, creosote, etc., may be utilized in the treatment of phthisis with advantage. It is, however, to be borne in mind that large doses are necessary to procure results, and that these preparations contain but a small proportion of cod-liver oil.

## CHAPTER XXXVII.

### SPECIFIC TREATMENT.

STRICTLY speaking, the term "specific" should only be applied to a remedy or preparation which has a proved selective curative effect on a certain disease. From this viewpoint we can state unequivocally that we have no specific remedy for tuberculosis in any of its clinical forms. We have no substance, drug, or preparation, which will cure or remove or ameliorate the symptoms in the vast majority of phthisical patients to the same degree as mercury or salvarsan is efficacious in syphilis, quinine in malaria or thyroid in myxedema. This is a fact which all thoughtful workers in the tuberculosis field acknowledge; even those who employ tuberculin extensively, and do not hesitate to call it specific treatment, say that it is only a good adjuvant to other therapeutic methods which should be tried in selected cases as long as a true specific is not available. Moreover, it appears that tuberculin only works in sanatoriums, where the patients are, in addition to the specific treatment, subjected to a rigorous hygienic and dietetic regime. It is distinctly stated that when the latter is lacking, tuberculin is of no avail.

It appears that the only justification for the use of the term specific when speaking of tuberculin treatment is the fact that this word has recently received a wider application and is now also used to designate remedies which are especially indicated and used in any particular disease.

The writer has given tuberculin therapy a fair trial in both his hospital and private practice and found it either altogether wanting in therapeutic effects when used in infinitesimally small doses, as is advised by most of its contemporary advocates, or decidedly harmful when given in substantial doses. This opinion is shared by most of those engaged in the treatment of tuberculosis, excepting such as have themselves discovered some tuberculin, or who are in charge of sanatoriums catering to well-to-do private patients. In the public sanatoriums in this country very little of tuberculin is used for therapeutic purposes. The vast majority of patients in these institutions are cared for by the old methods. It cannot be said that it is the cost which precludes the use of tuberculin in public institutions. Salvarsan is a really expensive drug but is used in all hospitals.

Our reasons for discarding tuberculin from the therapeutic armamentarium are the following:

**The Variety of Tuberculins.**—It is an old axiom in therapeutics that the larger the number of drugs recommended for any given disease, the less the chances of curing it with any of those mentioned as effica-

cious. Thus, we have only to consult the index of any standard materia medica and count the number of remedies recommended for typhoid fever, pneumonia, nephritis, gastritis, etc., and to compare it with the number mentioned as effective in myxedema, malaria, syphilis, valvular heart disease, etc., to be convinced that the axiom holds good. The large number of tuberculins alone should give us a strong hint that none of them is a specific, or will surely cure. I counted in one recent book forty-six varieties of tuberculins, and I could add almost as many which the author has not mentioned.

"We have no standard tuberculin," says William Charles White,<sup>1</sup> himself an advocate of tuberculin, "and furthermore we have no manufacturer who prepares the same strength twice. Consequently the dose of one tuberculin is no more the dose of another tuberculin than the dose of a sherry glass is the dose of a champagne glass. We have no method of testing the strength of a given tuberculin unless it is the biological one, and this is tedious, if it has to be used for every patient for every new supply of tuberculin. If, however, the tuberculin standard is at fault, what a vastly greater difference exists in the physicians who administer it. There are almost as many methods of dosage and administration as there are administrators. Each physician believes his method the best. Some have no method at all." It appears that for practical purposes we have no methods to weigh or measure the toxicity of tuberculins. Two preparations made by the identical method may differ very much if they are derived from different cultures, especially do they vary with the age of the culture.

All authors entitled to an opinion agree that the action of all tuberculins is the same. The preparations differ only as regards their strength, toxicity, capacity for absorption, etc. But inasmuch as the active element or substance of tuberculin has not yet been isolated, nor can the strength of a given preparation be measured, it appears that the differences which are known to exist between the various forms of tuberculin cannot be definitely ascertained. Salvarsan, strychnin, morphin, digitalis, or tetanus, and diphtheria antitoxin which could not be measured would hardly be used by medical men.

In general it may be stated that there are three varieties, or types of tuberculin:

1. *Old tuberculin*, consisting of the exotoxin—a glycerin extract containing the soluble products of the tubercle bacilli in the medium in which they have grown, glycerin, bouillon, extractives, etc. Though it should be mentioned that most investigators are of the opinion that there is no tuberculous exotoxin.

2. The *new tuberculins*, made up of the insoluble endoplasm of the bacilli and the poisons contained within them—endotoxins.

3. Those which consist in a mixture of both the above forms.

<sup>1</sup> Trans. Fifth Annual Conference Nat. Assn. Prev. Consumption, London, 1913, p. 70.

But when injected into the tuberculous human or animal body any tuberculin produces practically the same effect. On this nearly all agree, even those who maintain that only a certain variety of tuberculin should be used if therapeutic results are to be obtained.

**Action of Tuberculin.**—As was already stated (see p. 31), tuberculin is harmless when injected into a non-tuberculous body, and produces its toxic effects only in those who have suffered a tuberculous infection. But we do not know how it acts under these circumstances. Wolff-Eisner's tuberculolysin hypothesis is about the most plausible and the one accepted by most authors. But we have not as yet succeeded in isolating a specific tuberculous antibody, nor the tuberculolysin from the serum of infected animals.

At first sight it would appear that tuberculin is specific, considering that it acts only on infected organisms, but even this is not conclusive. It seems that the infected organism is not only hypersensitive to tuberculin, but to all foreign proteins. We can produce elevation of temperature, malaise, backache, nausea, etc., and even the local reaction, by the injection of any foreign protein into a tuberculous person. "Neither the local nor the general reaction is absolutely specific," says Baldwin,<sup>1</sup> himself using tuberculin extensively; "various nucleoproteins, yeast nuclein, bacterial proteids in general, and digestive products, such as albumoses, are capable of producing similar effects. Cinnamic acid, cantharidin, pilocarpin, and other alkaloids also act to some degree, although less as local irritants than general leukocyte stimulants." In my experience potassium iodid, and creosote, when given in large doses, may produce general and focal reactions not unlike those produced by tuberculin.

All efforts at producing partial or complete immunity with the administration of tuberculin in man or animals have utterly failed. Even Sahli, who urges tuberculin treatment, says that "tuberculin treatment has not the character of a true immunization, though it produces immunizatory effects in the organism."

That it is not necessarily the reaction which is effective therapeutically is clear when we consider that modern tuberculin treatment aims at eliminating entirely these reactions by the administration of infinitesimally small doses. The hope that the focal reactions, consisting in hyperemia at the site of the lesion and the surrounding tissues, may promote the healing of the lesion, cannot be seriously entertained by clinicians. Usually when the focal reaction is intense, it cannot be controlled and the congestion often produces renewed activity of the diseased process. Quiescent foci, calcareous particles, are "sleeping dogs" and should not be disturbed, as Sir James K. Fowler<sup>2</sup> says. The establishment of tuberculin tolerance, which some strive at, is no proof of healing; in fact it is usually short lived. Moreover, the tuberculin reaction is a very complex process and varies

<sup>1</sup> Osler's Modern Medicine, i, 308.

<sup>2</sup> Trans. Annual Conference Nat. Assn. Prev. Consumption, London, 1913, v, 93.



with the preparation used, the individual treated and also with the time it is administered. One day the patient is tolerant, the other he is badly affected with even a minimal dose.

There is no harm in administering most drugs in teaspoonfuls, tablespoonfuls, or measuring them with the point of a knife, as has been done for centuries. Patients have recovered with such inexact measures, some may have been harmed, but lethal doses are rarely given in this manner. But we cannot give a potent agent like tuberculin to a patient who needs all the vital energy he has, and more, in this manner, any more than we can give with impunity strychnin, morphin, digitalis, salvarsan, etc., without exact dosage. As long as we cannot measure the toxicity of tuberculin, we cannot administer it rationally and prevent sudden, and at times harmful, reactions which may appear when least expected.

**Experimental Evidence of the Lack of Therapeutic Effects of Tuberculin.**—Tuberculin as a therapeutic agent is based on results obtained in the laboratory through animal experimentation. It would be reasonable to exact that it should be efficacious in experimental tuberculosis in animals. But it is a fact that there is no record in medical literature that any investigator has succeeded in curing or benefiting a tuberculous animal with tuberculin treatment. In Robert Koch's writings at the time he introduced tuberculin we can find no clear-cut statement to the effect that he cured an animal with this agent. Klimmer, Lydia Rabinowitsch,<sup>1</sup> and others have recently tried small, very small doses, corresponding to those used at present in the treatment of human phthisis, but the tuberculous guinea-pigs and rabbits failed to improve. "No curative influence has been exercised by the tuberculin. The control animals lived sometimes longer than the treated animals. On the use of large doses the animals readily succumbed."

It has never been observed that the administration of tuberculin to tuberculous animals should promote healing of a tuberculous lesion, that cicatrization should be favored.

What has been observed, however, is that very often dormant tuberculous processes are activated after the administration of tuberculin. Bacilli which gave no trouble were released, "mobilized," producing a bacteremia, as was already mentioned (see p. 226).

Serologically, tuberculin has hardly ever shown its therapeutic value. Like other antigens, tuberculin stimulates the production of antibodies when inoculated into a tuberculous organism. But these antibodies cannot be considered true antituberculins because they do not neutralize tuberculin *in vitro*. We know that the antibodies produced by other toxins, as those of tetanus and diphtheria, neutralize the toxins of these infections *in vitro*, while the tuberculous antibodies do nothing of the kind. We can consequently see no theoretical or practical value in tuberculin from this viewpoint.

<sup>1</sup> Trans. Annual Conference Nat. Assn. Prev. Consumption, London, 1913, p. 44.

**Clinical Evidence.**—In a discussion on the merits of tuberculin treatment, Hector W. G. Mackenzie<sup>1</sup> said that “he should like to ask whether anyone has been able to obtain a cure of tuberculous ulcer, arising from the primary inoculation by means of tuberculin injections. He fears the answer must be in the negative.”

We arrive at the same conclusion when we consider the clinical evidence presented by the advocates of tuberculin treatment in phthisis. All effective medication has its indications, contra-indications, and limitations. True specific treatment is not free from these limitations, as is true of quinin, mercury, salvarsan, thyroids, etc. But the limitations in the range of usefulness of these drugs depend mainly, if not entirely, on the presence or absence of mixed infection, of pre-existing diseases, on the constitutional peculiarities of the patient and complicating diseases. In a clear-cut case of syphilis in the average patient salvarsan or mercury will produce evident curative effects; malarial fever will be abated by quinin, myxedema is relieved by thyroids, etc. But in the purest forms of tuberculosis, in acute miliary tuberculosis, tuberculin is powerless, which fact alone should arouse suspicion as to its specific qualities.

It appears to be a general rule in pathology, as has been pointed out by von Hansemann,<sup>2</sup> that diseases which are not at times spontaneously cured cannot be cured by any known therapeutic measure. Rabies is usually mentioned as an exception, but even this may only be prevented; once it has developed, it cannot be cured. Specific therapeutics aims at curing diseases which are not known to be cured spontaneously. But it has never been observed that a patient suffering from acute miliary tuberculosis should be cured, the few cases mentioned by Cornet are all very doubtful. Acute miliary tuberculosis is the purest form of the disease without mixed infection; the tubercle bacilli, though disseminated all over the body, are found in each place in small numbers and they do not produce avascular masses from which medication is excluded. It should be the crucial test for specific treatment. As a matter of fact, however, tuberculin is altogether powerless in acute miliary tuberculosis, as it is in all progressive cases of phthisis.

Good results are reported by those who have used it in glandular, osseous, and articular tuberculosis in children. But we have already mentioned that these have a strong natural tendency to heal spontaneously in the vast majority of cases (see p. 391). Even surgeons advise and practise conservative treatment.

In phthisis the ideal cases are said to be those in the incipient stage of the disease. But when we recall that a really incipient case is one which has “slight or no constitutional symptoms including particularly gastric or intestinal disturbances or rapid loss of weight; slight or no elevation of temperature or acceleration of pulse at any time during

<sup>1</sup> Trans. Annual Conference Nat. Assn. Prev. Consumption, London, 1913, p. 9.

<sup>2</sup> Berl. klin. Wehnschr., 1911, xlvii, 1.

the twenty-four hours," we are not surprised that many recover with tuberculin treatment. It has been found recently that in Germany, France, and England many of those who were certified as tuberculous and eligible for sanatoriums, were fit for military service. Instead of sending them to institutions, as has been the rule during times of peace, they were sent to the trenches and in the vast majority of cases they stood the hardships of war as well as other soldiers.

The reasons for this anomaly are various. Blömel claims that 80 per cent. of these cases were wrongly diagnosticated. But even such as showed the presence of tubercle bacilli in the sputum were found fit for military service. To my mind there are many cases of abortive tuberculosis which under ordinary circumstances pass as chronic phthisis and any form of treatment gets the credit for the cure. Tuberculin evidently gets its share of credit.

**Lack of Reliable Statistics of the Efficacy of Tuberculin.**—To prove its therapeutic efficacy, a specific must produce results in a larger proportion of cases of phthisis than is observed with the older methods of treatment. This has not been shown. In fact there are no reliable statistics of large series of cases available. In their book on tuberculin treatment, Riviere and Morland state that they decided to give no statistics of results of tuberculin treatment because they consider figures of questionable value. Sahli also gives no statistics, while the figures compiled by Brown in Klebs's book show clearly that there is no difference in results between the group treated with as compared with that treated without tuberculin. Statistics of ultimate results are not available at all.

**Dosage.**—It would be pretty bad for physicians, and for patients, if there was such a disagreement as to the dose of any potent remedy, especially if it was not known which quantity of the remedy is likely to be harmful. The initial dose ranges between 1 mg., recommended by Bandelier and Röpke to 0.0000005 mg., recommended by Philippi. Between these two extremes, various authors recommend intermediate quantities, each one claiming that his standard is best, or, what is of more importance, the safest. Still, with such uncertainty as to dosage, many authors make tables of dosage, and iron-clad rules as to gradual increase in the dose, and the final dose, some using logarithmic tables for their calculations, as if they were dealing with an exact science.

The fact is that there is no mystery about the technic of administration of tuberculin, and no knowledge of higher mathematics is necessary to make the various dilutions properly. Many pharmaceutical houses sell tuberculin in proper dilutions ready for use. But those who want to make their own dilutions can do it easily.

All that is necessary is six or ten amber-colored bottles of 10 or 20 c.c. capacity each. They are to be clean and properly sterilized. A larger bottle containing the diluent (sterilized, or distilled water containing 0.8 per cent. of sodium chloride and 0.5 per cent. of carbolic acid) should be at hand. Each of the small, colored bottles is to be

filled with 9 c.c. of the diluent and marked with numbers, I, II, III, IV, V, VI, etc., respectively. Now take 1 c.c. of tuberculin and drop it into bottle No. I and shake it well. It now contains a 10 per cent. solution of tuberculin, so that a syringeful, with a capacity of 1 c.c., contains 0.1 c.c. of tuberculin, or 100 c.mm.

When we take 1 c.c. from bottle No. I and drop it into bottle No. II, we get a solution containing 1 per cent. of tuberculin; one syringeful contains 10 c.mm. of tuberculin. Repeating the process, dropping 1 c.c. from bottle No. II into bottle No. III, the latter will contain a 1 to 1000 dilution; 1 c.c. equals 1 c.mm. of tuberculin; bottle No. IV, a 1 to 10,000 dilution; bottle No. V, a 1 to 100,000 dilution; and bottle No. VI, a 1 to 1,000,000 dilution, so that a syringeful will contain a dose of 0.001 c.mm. of tuberculin. These dilution may be carried further and the dose, which should always be small, if administered at all, may be infinitesimally so.

If given for its psychic effects, which is in fact done at present by most who use this agent, it is advisable to have ten bottles and that the first injection should be made from bottle No. X. If the patient is impressed by the treatment, he will "react" at least with 0.3° to 0.5° F., which should satisfy any one who is looking for a "mild reaction."

Moreover, there is no difficulty in administering properly a series of ascending doses of tuberculin, and no higher mathematics is necessary for its successful accomplishment. Taking the first injection as a unit, we may increase the next injection by one-fourth or one-half. Thus, supposing we have used at first the dilution in bottle No. X containing 0.0000001 c.mm. of tuberculin per cubic centimeter, we inject but one-third or one-half of the contents of the syringe. The reaction is not likely to be severe, and we may one or two days later increase it to one-half or two-thirds of the contents of the syringe. In this manner we may proceed till we reach bottle No. VI, when the injection of a syringeful will give a dose of 0.001 cm. It is not advisable to give larger doses if we want to make sure that the patient is not harmed. But if there is any reaction the injections should be stopped promptly.

**Utility of Tuberculin Treatment.**—It cannot, however, be denied that some good results have been obtained with tuberculin treatment. Whether they could not be obtained with other methods in those cases is another question. Thus, E. Rist<sup>1</sup> says, "for my part, I have never seen a patient doing well under tuberculin without remaining in doubt whether he would not have done as well without tuberculin. Nor have I met with cases where the influence of tuberculin was so strikingly favorable that I could feel justified in letting them abandon the classical treatment and rely on tuberculin alone." Sir James K. Fowler says: "The tuberculin did not favorably influence the course

<sup>1</sup> Paris Médical, 1913, iv, 241.

of the disease in the majority of cases; in some cases the effects were detrimental; and even in stationary and improved cases it was difficult to ascribe any distinct improvement to the injections which might not have been equally attained under the treatment ordinarily employed in the Brompton Hospital."

In the extensive *Handbook on Tuberculosis*, A. Schröder<sup>1</sup> shows that "It has been established that in institutions for the treatment of tuberculosis in which only general treatment is applied, the lasting results obtained are not inferior to those reported from institutions in which, in addition to the general treatment, so-called specifics are administered."

Good results are obtained with tuberculin only when carefully administered in sanatoriums, with cases in the incipient stage, with but slight lesions, most of which are spontaneously curable. Although, according to Brown, at the Adirondack Cottage Sanatorium no selection is exercised—the patients are allowed to elect tuberculin treatment. In private practice, as well as in most tuberculosis clinics in cities in this country, attempts with tuberculin have failed, evidently because the good surroundings, the fresh air, the proper food, regulation of rest and exercise were of more importance than the tuberculin. When we consider further that even the most ardent advocates of tuberculin state that only cases without fever, pursuing a slow course, showing no tendency to progress, but manifesting a strong tendency to fibrosis, are suitable for the treatment, it is clear that tuberculin is a remedy for those forms of phthisis which are spontaneously curable.

**Psychic Effects.**—We have seen that the tuberculous patient is very amenable to suggestion (see p. 236) and we have pointed out that in a certain class of cases tuberculin produces excellent results for this reason. On this point a large number of physicians agree, and they continue to administer tuberculin because of its psychic effects, although they may as well administer distilled water hypodermically and obtain the same results. To keep nervous, irritable, fretful patients for months, or even for years, is a difficult matter; often it is an impossible affair. Something must be done in addition to the rest, fresh air, milk, and eggs; of which he believes he knows as much as his doctor. Such patients, when given tuberculin, told to watch out for reactions, to record in detail the symptoms produced by each ascending or descending dose on a specially prepared blank, are often very much encouraged.

This view of the psychic action of tuberculin is entertained by most authoritative physicians who use this agent extensively. Thus, Lawson Brown,<sup>2</sup> who has done so much to popularize tuberculin in this country, says that only poor results can be expected when it is given "in cold blood." He believes "its value can be greatly enhanced

<sup>1</sup> Brauer, Schröder, and Blumenfeld's *Handbuch der Tuberkulose*, 1915, ii, 3.

<sup>2</sup> *Amer. Jour. Med. Sci.*, 1912, cxliv, 524.

when the administrator has implicit faith in its curative properties and imparts that faith to his patients." Another significant reason for using tuberculin treatment according to Brown "is the closer relationship that such treatment establishes between patient and physician. I must confess that I find it difficult to bring a patient to my office twice a week for months and discuss symptoms and fears, one of which gradually grows less while the other is often replaced by more or less indifference, borne of familiarity. When, however, I give this patient tuberculin, he and I can discuss his case in detail twice a week and I am able to discover slight but important changes in his condition, to check imprudence, and to change needless timidity into confidence in his ability to order aright his life."

But similar results have been obtained by Mathieu and Dobrovici with "antiphymose" as was already detailed (see p. 443). In valvular heart disease, syphilis, myxedema, etc., this does not work.

I believe that I am safe in saying that as a rule tuberculin treatment is only efficacious in intelligent patients who are under the impression that they have mastered the theoretical aspects of infection and immunity and of specific therapy from reading popular books and articles on tuberculosis. In fact, in my experience, uneducated patients hardly ever improve under tuberculin treatment because they cannot understand the benefit of fever, malaise, pain in the limbs, nausea, debility, etc. On the other hand, intelligent patients look forward to the reaction as an indication that the tuberculin is "working on their system" and they often improve, provided infinitesimally small doses have been given.

There is no agreement among authorities as to what constitutes a "reaction" during tuberculin treatment. "All physicians are agreed that severe reactions are harmful to the patient as a general rule," say Archer W. R. Cochrane and Cuthbert A. Sprawson,<sup>1</sup> "but there is still considerable difference of opinion between those who like their course to progress without any reactions at all, and those who prefer mild reactions as a routine. Again, opinion varies as to what constitutes a mild reaction. In dealing with those otherwise running a normal temperature the limit by some has been fixed at 100.4° F., and reactions thereto are disregarded; that is to say these physicians will increase the next dose if the last dose has not given a reaction over 100.4° F." But these authors consider this limit too high or dangerous, and are satisfied with a rise to 99.2° F. and call it a reaction. In other words, "the timid, or no-reaction school," treat only afebrile cases. They should meet with immense success, because this class of patients recover spontaneously or with any kind of treatment.

**Dangers of Tuberculin Treatment.**—Since the first use of tuberculin as a therapeutic agent, it has been recognized that it is capable of

<sup>1</sup> A Guide to the Use of Tuberculin, London, 1915, p. 60.

doing irreparable damage when imprudently administered. Virchow found that it produced rapid disintegration of the tuberculous tissues in the lungs, caseous pneumonia, and at times eruption of miliary tubercles. More recent investigations have shown that it often mobilizes the bacilli and thus may favor metastatic auto-infection. In fact, if phthisis was not a manifestation of immunity, disastrous results from this cause would be very frequent. It has also been observed that patients taking tuberculin for a long time are likely to develop nephritis. To be sure, with infinitesimally small doses the likelihood of such complications is reduced to a minimum, but the most experienced administrator is often surprised by unexpected reactions. I have seen such results repeatedly; mostly when tuberculin was administered by such as were not skilled in handling this potent agent, but also at times in patients who were treated by very skillful physicians.

Producing hyperemia of the affected lung area, tuberculin at times is effective in inducing pulmonary hemorrhage. When large doses were used this was very frequently observed and reported by Fränkel, Rumpf, Stricker, and many others. "Since small doses have been used," says J. Sörgo,<sup>1</sup> "with a view of avoiding strong reactions, hemoptysis is only rarely observed after the administration of tuberculin. At times small hemoptyses are seen, especially streaky sputum, but copious hemorrhages are rare. For this reason it is agreed that a tendency to hemoptysis is not altogether a contra-indication to tuberculin treatment, provided strong reactions are avoided." But, as we already mentioned this is not possible in every case. All who administer tuberculin for therapeutic purposes stop the treatment as soon as bleeding makes its appearance.

The general practitioner should not use tuberculin at all. He can obtain the same results by the judicious use of drugs without incurring any risk. Even psychotherapy of the kind applied by those who administer tuberculin can easily be practised with medication, as was shown in Chapter XXVIII.

<sup>1</sup> Brauer, Schröder, and Blumenfeld's *Handbuch der Tuberkulose*, 1914, ii, 255.

## CHAPTER XXXVIII.

### SYMPTOMATIC TREATMENT.

**Cough.**—To many patients the cough is the disease and they are under the impression that all they need for a speedy recovery is to be rid of this annoying and painful symptom. In its treatment some points are to be borne in mind: In most cases cough is decidedly conservative—a purposeful reflex act; it removes the secretions from the respiratory passages which, if retained, might act like foreign bodies or produce toxic effects. But, on the other hand, cough often disturbs the affected tissues which need rest, if cicatrization is to occur, or it may be responsible for hemoptysis, pneumothorax, etc. Usually these conflicting principles can be reconciled by appropriate treatment.

Cough can be prevented or ameliorated by simple measures in a large proportion of cases. Atmospheric purity contributes considerably toward a reduction in its frequency and severity. Outdoor life and good ventilation of the room inhabited by the patient meet this indication. Mouth-breathing is a cause of excessive coughing in many cases, and some get fits of coughing when suddenly changing from a warm into a cold atmosphere, or the reverse. In steam-heated rooms, in which the air is usually dry, cough is more frequent than in rooms in which the air contains a proper amount of moisture.

In advanced cases with secreting cavities, the cough may be influenced by posture; reclining on one side expectoration is facilitated, while lying on the other side brings about violent fits of coughing. Patients soon find out which position gives them relief and recline accordingly. These patients may only cough during the morning hours and thus empty their cavities of the secretions which have accumulated during the night, while during the day there is but little cough. They need no treatment for this symptom.

It will be observed that some phthisical patients who sleep well during the night cough more during the day than those who cough more or less during the night. The administration of large doses of opiates during the evening may gain relief in sleep, but also result in miserable hours during the following day. This is to be remembered when administering opiates to tuberculous patients.

**Psychotherapy of Cough.**—It is a noteworthy fact that the cough is greatly influenced by the psychic state of the patient. Persons with an irritable nervous system, the hysterical, emotional and neurasthenic, cough more than the dull, the phlegmatic and apathetic. Some cough while in the house and are relieved as soon as they go out into the



open air, while in others the cough increases as soon as the window is opened, or when they go out into the open air on a cold day. This last class of patients is very difficult to manage.

Other psychic influences are seen in patients who usually cough excessively but cease when in agreeable company, or are intensely interested in something, etc. I have practically stopped unproductive cough in many patients by threatening them with expulsion from the hospital if they did not cease annoying their fellow-sufferers in the ward. Lonesomeness, and also insomnia, are often responsible for excessive cough and should be treated according to indications. In sanatoriums the influence of example is often very good: The patient sees others control their cough and attempts to do likewise and is often surprised at his success.

The patients can, within certain limits, control their cough, as Galen pointed out more than seventeen centuries ago, and Dettweiler has shown that this symptom can be "disciplined." Even when the cough is productive of considerable quantities of sputum, the patient is to be instructed that he need not expel it all at once; that if he succeeds in suppressing it for some time, the accumulated sputum will later be brought out with little effort. During the morning hours patients often make strong efforts to clear their chests. But if they should wait till after breakfast they may find that the sputum comes up easily. "Cough induces cough," says Penzoldt,<sup>1</sup> and for this reason patients are to be warned against giving in to the first tickling of the throat. The great struggle will only be during the first two or three days. Meeting with success, patients become convinced of their own powers to suppress or control this symptom.

But patients must be warned in this connection against swallowing their sputum—"spitting into their own stomachs." Controlling does not mean entirely suppressing expectoration as women and some men are apt to do. The dangers of the habit are to be explained in detail to the patient.

I cannot agree with those who prohibit smoking to tuberculous patients indiscriminately. To be sure those who are not accustomed to tobacco often cough when near a person who smokes. But many habitual smokers are greatly relieved by a cigar or cigarette. Our advice should be in accordance with the experience of the individual patient.

Many home remedies are very often efficacious in relieving cough. Thus, equal parts of boiled milk and honey or glycerin, with or without a flavoring agent, may be of great use in stopping an annoying cough. An excellent remedy is the application of a small mustard leaf, or blister over the seat of the lesion. It may be repeated from time to time. The fact that it works by psychic suggestion should not deter us from using it, so long as the patient gets relief.

<sup>1</sup> Handbuch der Therapie, 1910, iii, 249.

**Medicinal Treatment.**—After all the cases in which the cough may be controlled, or made bearable by simple methods, are discounted, there remain a large number who must be given sedatives to control this symptom. In the incipient stage these remedies are only rarely called for, and then only for a short time. But in advanced cases the indications for sedatives become more and more urgent. As Penzoldt says, the more progressive the disease, and the less the chances of ultimate recovery, the more the charity of morphine is to be dispensed to the unfortunate sufferer.

In my experience, many cases in the incipient and moderately advanced stages of the disease are immensely relieved by creosote and its derivatives. The method of administration is given elsewhere. In those in whom internal administration does not relieve the cough, we may try the effects of inhalation of creosote, menthol, eucalyptol, tincture of benzoin, etc. The following is as good as any that has been recommended:

R̄—Creosoti,  
 Acidi carbolicī,  
 Spir. chloroformi . . . . . āā ʒiv 15.0  
 M. S.—Ten to twenty drops in an inhaler, to be used for fifteen minutes at a time.

Failing with these simple remedies we must resort to anodynes in case the cough is frequent, violent, paroxysmal, or disturbs the patient's comfort or sleep. Of these, cannabis indicæ is the least harmful and should be given the first trial. The extract may be given in doses of  $\frac{1}{4}$  grain in pill or tablet form several times a day. In spasmodic cough it may be combined with hyosciamus or gelsemium. The following may be used to great advantage:

R̄—Extracti cannabis indicæ . . . . . gr. vj 0.4  
 Extracti hyosciami . . . . . gr. xij 0.8  
 M. ft. pil. No. xxiv.  
 S.—One pill four to six times a day.

R̄—Extracti cannabis indicæ fl.,  
 Extracti gelsemii fl. . . . . āā ʒij 8.0  
 Syr. acaciæ . . . . . ʒj 30.0  
 Aquæ menthæ piper. . . . . ad ʒiv 120.0  
 M. S.—One teaspoonful four times a day.

In many cases nothing but opiates gives relief. But in incipient cases opium and its derivatives are to be avoided because it may have to be continued for long periods and, in hopeful cases, the danger of habit formation is not negligible. In addition opium deranges the digestive functions, produces anorexia and constipation, slows the frequency and the amplitude of the respiratory movements, and favors stagnation of the secretions in the respiratory passages. A dose of Dover's powder may be given in the evening now and then with a view of controlling the cough during the night, but to continue the administration of opium in any form for any length of time is dangerous.

Of the many opiates, codein, which is ten to twelve times less toxic than morphine, is to be preferred. It may be given in tablet form in doses of  $\frac{1}{8}$  to  $\frac{1}{4}$  grain, and in advanced cases even in much larger doses several times a day; or it may be added to any other medication that is being administered. Thus I quite often add it to creosote medication:

R—Guaiacolis carbonatis . . . . .	ʒiiss	10.0
Strychninæ sulphatis . . . . .	gr. j	0.06
Arsenici trioxidi . . . . .	gr. j	0.06
Codeinæ phosphatis . . . . .	gr. viij	0.5

M. ft capsul. No. 1.

S.—One capsule three times a day after meals.

R—Codeinæ sulphatis . . . . .	gr. iv	0.3
Extracti cannabis indicæ . . . . .	gr. vj	0.4
Extracti belladonnæ . . . . .	gr. iij	0.2
Extracti glycyrrhizæ . . . . .	gr. xij	0.8

M. ft. pilullæ No. xii.

S.—One pill at night.

In most cases in which sedatives must be given for a considerable time the dose must soon be increased because after a few weeks the effects on the cough are diminished. Instead of increasing the dose, we may do better by changing one for some other derivative of opium. Heroin may be given in doses of  $\frac{1}{24}$  to  $\frac{1}{8}$  grain according to indications. It does not constipate and when there is dyspnea it is the best palliative remedy. Dionin is another of these preparations and, when insomnia is a troublesome feature, it is even better than the above. Not many cases of habituation to dionin have been reported, but it is more apt to cause constipation than codein or heroin. The two last mentioned preparations do not interfere with the expectoration of sputum; some even maintain that they assist in its expulsion. Whenever feasible these narcotics are not to be given after midnight in order to avoid headache and debility during the morning hours.

The emetic cough is a very difficult symptom to control in some cases. I have seen some in whom it was responsible for a bad turn in an otherwise favorable case. Rarely, no food can be retained. Most can be relieved by avoiding heavy meals—taking several small meals during the day. The patient should recline in bed immediately after meals and avoid any exertion and even speaking. But at times we must resort to medication. Some have reported good results from several drops of chloroform well diluted, or from bromoform. I have had cases in which only cocain administered before meals was effective in retaining nourishment in the stomach. The following prescription of Albert Robin may have to be resorted to:

R—Cocain hydrochloratis . . . . .	gr. j	0.06
Codein sulphatis . . . . .	gr. j	0.06
Aquæ chloroformi . . . . .	ʒij	60.0
Aquæ . . . . .	ʒiv	120.0

M. S.—Tablespoonful after meals.

**Expectoration.**—In the average case of phthisis expectoration is a salutary phenomenon, removing, as it does, foreign, often toxic, material from the respiratory passages. At times it becomes excessive and annoying, but it should never be suppressed. In some cases with extensive excavations the amount of sputum brought up may be controlled within limits by posture. We advise our patients to recline in certain positions which favor the expulsion of sputum and thus empty the cavities of their contents. Relief may thus be obtained for the rest of the day. In cases in which the sputum is fetid—rare in phthisis—antiseptic inhalations may be tried. Creosote, iodine, menthol, eucalyptol, turpentine, etc., may be inhaled through an inhaler or simply dropped in hot water and inhaled.

Very often patients complain that they feel heavy on the chest and that if they could only bring up sputum they are confident that they would be relieved. Many drugs have been used for this purpose, especially the so-called expectorant remedies. It seems that all that is usually attained is a disordered stomach.

It appears from recent pharmacological investigation that there are no drugs which, when given in small doses, will induce more abundant secretion into the respiratory passages, stimulate the cilia of the bronchial mucous membrane to bring out secretions, or render tenacious secretions more easily movable from the bronchial walls to which they adhere. J. L. Miller<sup>1</sup> found that ammonium carbonate and ammonium chloride, and the emetic group of expectorants, as apomorphin and ipecac, when given in sufficiently large doses to animals, increase the bronchial secretion. Ammonia salts per os, in moderate doses equivalent to 2 mg. in an adult man, do not increase bronchial secretions in the dog. Apomorphin and emetin, when given to dogs in doses considerably greater than the ordinary therapeutic dose for man, do not excite increased bronchial secretion.

It is therefore absurd to give nauseating potions of ammonium salts, senega, ipecac, morphin, etc. All we may succeed in doing is to disorder the stomach, but the secretion, in the respiratory passages remains unaffected.

**Fever.**—Fever is an indication of active, often progressive phthisis, unless due to some complication. Its continued presence proves conclusively that the disease is spreading, even if the physical signs remain unaltered. It is at times neglected or overlooked because, unlike fever in other diseases, the patient in spite of a temperature of over 100° F. may feel quite comfortable, have a good appetite, and even gain in weight. But the entire future of the patient may depend on the treatment of the fever; neglecting mild febrile attacks means an invitation for chronic prolonged fever with lessened chances of recovery.

During the initial stages of the disease fever demands rest in bed, not so much as a cure but as a preventative against the extension of

<sup>1</sup> Amer. Jour. Med. Sci., 1914, cxviii, 469.

the process in the lung. It is remarkable that in many cases the fever abates within a few days or a week only through an improvement in the hygienic conditions and the diet of the patient, and placing him in a light and well-ventilated room. It is unfortunate that very few patients are willing to submit to perfect rest at this stage, claiming that they are not sick.

There are many advanced cases of phthisis with quite extensive lesions in which there is a daily rise in the temperature of 1 to 1.5° F., but the patients feel quite well and are even able to pursue their vocations. They need no active treatment because they have become habituated to the subfebrile temperature which may be regarded as their normal condition. In this class of cases it is only necessary to take steps to reduce the temperature when the patient is clearly suffering as a result of it; when the fever produces symptoms such as anorexia, restlessness, irritability, insomnia, etc.; or when he is losing in weight. I have observed many cases in which fever was due to overfeeding, and a reduction in the quantity of food has promptly brought the temperature down to normal.

A sudden rise in the temperature in the course of chronic phthisis may be due either to an extension of the lesion, a new pneumonic process in a hitherto unaffected part of the lung, or to some complication. The former demand rest in bed till the temperature comes down to normal; in the latter the indications are in accordance with the pathological conditions which present themselves.

Patients are apt to attribute an attack of fever to "indigestion," but in my experience acute gastritis is a rather infrequent cause of pyrexia in phthisis, though a dose of calomel at times relieves an evanescent febrile attack. More often fever lasting several days is due to influenza or tonsillitis. In hospital practice there is at times seen an actual epidemic of these diseases, most of the patients in the ward are attacked during a period of a couple of weeks. The treatment is rest in bed and some antipyretic, like antipyrin, quinin, aspirin, etc. Complicating pleurisy, with or without effusion, may be the cause of a rise in temperature. In some women premenstrual or menstrual fever demands rest in bed periodically for a few days. The instability of the temperature in phthisis, which has been discussed in a previous chapter, is responsible for many febrile attacks. Any physical or mental exertion, worry, grief, and anxiety may raise the temperature several degrees. Prophylactic and curative action is indicated along these lines.

The fever accompanying active phthisis demands active treatment. The main aim should be to remove it, or to prevent its occurrence. If we fail in this, we fail in our efforts at relieving the patient. It may very often be prevented by putting a patient to bed at the very first indication of a tendency to hyperthermia from any cause. Indeed, the neglect of mild febrile attacks is very often responsible for prolonged and even fatal fever.

In high continuous fever perfect rest is indicated, preferably in the open air, or in a room with wide open windows, as has already been detailed in Chapter XXXI. The patient is to be treated as though he is suffering from an acute disease, like typhoid or pneumonia. It is often surprising to note the prompt improvement after a rest in bed for a few days. Patients with a temperature at a high level for several months are often difficult to manage. When accompanied, as it usually is, by progressive loss of appetite, weight, and strength, they become discouraged and rebel against the prolonged and strict confinement. In such cases, provided the temperature is below 101° F., the experiment may be made of permitting them to leave the bed and get out in the open, resting on a reclining chair for a few hours during the day. The best hours are before or around midday, when the temperature is usually at its lowest; but any other time may be chosen under the guidance of the thermometer. In hectic cases the temperature is usually at its lowest in the morning and the patient may be allowed to leave his bed at that time. I have seen many patients, who did badly for weeks, improve when allowed to remain in the upright or semiupright position for several hours a day. But care and circumspection are to be exercised while applying this treatment.

Some patients may be sent to the country and the change is at times effective in reducing the temperature when everything else has failed. But this is not available to patients who have not the means to leave accompanied by an attendant. Many authorities state that a mountainous climate is to be preferred for this purpose, but in my experience any change may do just as well.

It is deplorable that public sanatoriums do not admit febrile cases. Great service could be rendered by removing the patient for several weeks, during the period of fever, to better surroundings, giving him an opportunity to rest without interference by well-meaning, but often ill-guided, relatives and friends. I have often felt that cases under my care could be saved if sanatoriums were managed along hospital lines, admitting patients during acute exacerbations in the places which are now filled with patients whose condition is such that they would do well in any healthy surroundings which can be obtained in the average home.

Hydrotherapeutic measures have not been found satisfactory in the treatment of fever in phthisis. The use of ice, or of cold sponging, or bathing, although possibly of temporary benefit, is contra-indicated in most cases because they are apt to depress the patient. The most that can be done is to give a warm or tepid bath once or twice a week for the purpose of cleansing the body, but care is to be taken not to subject him to overexertion while going and coming from the tub. The fact that hydrotherapeutic methods have been given up in nearly all sanatoriums is sufficient proof that they have not been beneficial; in fact that they were harmful.

Artificial pneumothorax is an excellent radical measure against

tuberculous fever in appropriate cases. This will be discussed in Chapter XXXIX.

**Antipyretic Medication.**—Antipyretic drugs should only exceptionally be used in phthisis. In the first place tuberculous patients do not, as a rule, suffer from the pyrexia to the same extent as patients with typhoid fever, pneumonia, etc., and a reduction in the temperature does not necessarily give the relief which the patient anticipates. It is not the fever, excepting hyperpyrexia, which is dangerous, but the activity of the tuberculous process, and as long as only the former is influenced, the patient is not materially benefited.

The action of antipyretic drugs is ephemeral and deceptive, often accompanied by profuse perspiration which is enervating; and by digestive disturbances. Large and frequently repeated doses are necessary for weeks in the usual cases and their action on the heart, which is not salutary, often leads to collapse.

But when the fever is accompanied by headache, backache, and debility, one of the coal-tar antipyretics may give comfort with or without reducing the temperature. Acetanilid is to be avoided for well-known reasons. Phenacetin acts too quickly and produces profuse sweating. Antipyrin, or better, pyramidon may be used in 5- to 10-grain doses, combined with caffeine. Patients may stand the fever without complaining much, but in septic cases they abhor the chills which are apt to occur before the onset of the pyrexia. The best treatment is to place the patient in bed a few hours before the appearance of the chill, cover him well, and give him a drink of hot lemonade, tea, or whisky and, in severe cases, a dose of pyramidon. The chill may in this manner not be prevented completely, but it is rendered bearable. On the whole, antipyretic medication is to be administered an hour or so before the highest temperature is expected, varying with each case. Quinin should be given, if at all, five to six hours before the maximum temperature is expected, while pyramidon, antipyrin, aspirin, etc., require but two to three hours. When the fever has declined medication should not be continued, otherwise collapse may occur.

The salicylates are often very good in these cases, especially in the chronic hectic fever of consumption. The old prescription of sodium salicylate and arsenous acid (sod. salicyl., 10; acid. arsenicosi, 0.01; ft. pil. no. 100; S., five to ten pills three times a day after meals) is very good. But I have found that 7 to 10 grains of aspirin and  $\frac{1}{150}$  gr. of arsenic in capsule three times a day is better. It is less likely to disturb digestion. But in patients showing a tendency to hemoptysis the salicylates are to be avoided. Pyramidon is best for this class of patients.

An excellent remedy for fever in tuberculosis is guaiacol painted with a camel-hair brush on the skin in 7- to 15-drop doses and covered air tight. The temperature drops sometimes within one hour. It is best to rub into the skin of the thorax a teaspoonful of a 10 per cent.

guaiacol-vaselin ointment two or three times a day. It must be mentioned that collapse has been observed in some cases after the application of guaiacol.

**Nightsweats.**—No other symptom of chronic phthisis is more discouraging and enervating than nightsweats and their relief is of immense importance. It seems that in the vast majority of cases they can be prevented without the use of medication and many physicians state that with careful prophylaxis they have not used any drugs for this symptom for years.

Open-air treatment is the best preventative of nightsweats. Sleeping in a cold room with sufficient but not excessive covering must be enjoined. It is also good to give the patient before retiring a glass of cold milk with three or four teaspoonfuls of cognac to prevent the rapid sinking of the pulse rate. In some cases a roll with butter may serve the same purpose. Some cases may be relieved by noting the time of the beginning of the sweating, and waking the patient a few minutes before and giving him an ounce of whisky. For private patients an alarm clock may be used for the purpose. This method, recommended by William Porter,<sup>1</sup> should be tried in all obstinate cases.

In cases in which these simple measures do not succeed, the sulphate of atropin in doses of  $\frac{1}{100}$  grain, given in tablet form about seven o'clock in the evening, may give complete relief. Agaricin is also good in doses of  $\frac{1}{20}$  grain, but it acts more slowly and must be administered about six hours before the sweating is expected. It often produces gastro-intestinal disturbances, especially diarrhea, and should be combined with an opiate—Dover's powder in 3- to 5-grain doses. It is to be remembered that no remedy retains its power over this symptom for a long time, and after one ceases to act, we may try another.

Friction of the skin with tepid water, or vinegar or alcohol and water, or a 3 per cent. lysol solution, may give relief.

**Hemoptysis.**—The prophylaxis of hemoptysis cannot be considered an easy matter despite the fact that we speak so much about the predisposing and exciting factors of pulmonary hemorrhage. Patients with really initial hemoptyses always consult us after the accident has occurred; and while many of the hemorrhages occurring during the course of phthisis have some exciting cause—overexertion, excitement, acute exacerbations of the process, etc.—behind them, there are just as many in which the patient had been at perfect rest physically and psychically. In fact, many of the copious and fatal hemorrhages occur during the night, when the patients were asleep. However, all patients with pulmonary tuberculosis are to be told in advance that there is less danger in blood-spitting than is generally believed. We would thus avoid the psychic depression which is so often an accompaniment of hemoptysis. Women may be told

<sup>1</sup> International Clinics, Sixteenth Series, 1906, iv, 77.



that in the average case of hemoptysis there is no more danger than in the loss of blood during the menstrual period.

Not all cases of hemoptysis require the same treatment; individualization is required here, just as in most other pathological conditions. The vast majority of hemorrhages are insignificant, and if we only place the patient at rest in bed, and quiet him by an assurance that there is little danger, the bleeding will cease sooner or later and the underlying process in the lung pursues its course uninfluenced by the accident. This is true of streaky sputum, which often terrorizes a patient to the same extent as a copious hemorrhage. But when the blood brought up is bright red, even if only a few mouthfuls, the matter is to be taken more seriously because these small hemorrhages are at times the precursors of repeated and copious, though rarely uncontrollable, hemorrhages. Immediate and absolute rest is to be enjoined and strong measures taken to stop the bleeding.

The patient is put to bed, but not in the traditional prone position. The blood and sputum must be evacuated from the respiratory passages with ease and this can only be done when the patient is in the semisitting position. In this manner nourishment and medication can be administered without unduly disturbing the patient, expectoration is facilitated and, in copious hemorrhages, atelectasis of the posterior parts of the lung is prevented. With a view of keeping a patient at perfect rest an ice-bag is to be applied to the chest. We know of no other value to this time-honored procedure. The patient is to be warned against any motion of the body and even speaking is prohibited. Only after several days without any blood in the sputum may the patient be permitted to assume the upright position.

The therapeutic indications to be met in addition to rest are: Prevention of excessive cough and expectoration; increasing the coagulability of the blood; and immobilization of the bleeding lung.

**Morphin.**—To allay excitement, procure rest, and thus prevent excessive cough, there is no better remedy than a hypodermic injection of morphin. We must bear in mind that we are in the presence of a conflicting situation. On the one hand, we must see to it that the effused blood in the bronchial tree should be removed; on the other hand, the strong expiratory efforts necessary to accomplish the expulsion of the blood and clots are accompanied by an increase in the pressure in the pulmonary circulation and, with their removal, the thrombi which plug the bleeding vessel are dislodged and thus renewed bleeding is likely to occur. Morphin meets but one of these indications: It depresses the cough centre, diminishes the frequency and amplitude of the respiratory movements and quiets the mental state of the patient. Some have even found that morphin increases the coagulability of the blood. But after all it has its dangers. When given to excess, as is often done, it depresses the respiratory centre, paralyzes the sensibility of the bronchial mucous membrane and thus interferes with the expulsion of the blood and clots. Aspiration

pneumonia may thus result in cases in which it is more successful as a hemostatic than is desirable.

For this reason morphin is to be used with great care and circumspection. Finding the patient excited and in agony, we inject hypodermically  $\frac{1}{4}$  grain of morphin for its general and local effects. If the bleeding does not stop within an hour the morphin should not be repeated, but other means are to be taken to control the hemorrhage.

**Emetin.**—In former time emetics were given in hemoptysis and excellent results were reported because with the vomiting the effused blood in the bronchi was also expelled preventing asphyxiation and also because the nauseous feeling reduced the blood-pressure perceptibly. Following Trousseau's suggestion, large doses of ipecac were given for this purpose. But we now have in emetin an excellent substitute for the nauseous ipecac. It acts as a hemostatic when many other agents have failed. I have used it in  $\frac{3}{4}$ -grain doses, repeated three to five times a day, with satisfaction. The simplest way of administration in these cases is hypodermically. Either the tablets or the ampoules, which many pharmaceutical houses prepare, may be used for the purpose.

**Salt.**—Another ancient remedy for copious hemorrhage is the administration of table salt. Formerly it was thought that because it acts as an emetic, and thus depresses the blood-pressure, it is of use in hemoptysis. But we now know that its *modus operandi* is different. Von den Velden<sup>1</sup> has proved that in man, swallowing 5 to 15 grams of table salt increases the coagulability of the blood within five minutes. Within one hour the coagulability returns to its former intensity. Sodium bromide has nearly the same effect. For this reason the administration of 5 to 10 grams of table salt or 3 grams of sodium bromide three to four times a day may prove of immense value in hemoptysis. In very nervous patients the bromide is to be preferred.

More recently salt has been administered intravenously in isotonic solution as recommended by Hans Müller.<sup>2</sup> Ten to 50 c.c. of a 10 per cent. solution of sodium chlorid, sterilized and heated to the body temperature, is injected into the median basilic vein, great care being taken not to drop any of the solution into the subcutaneous tissue which is likely to cause intense pain. I have tried this treatment and found it satisfactory in many cases.

**Tying the Extremities.**—The coagulability of the blood is also increased by tying up the blood in the extremities. A constricting band or a tourniquet is tied around the arm and the hip; two or three of the extremities are tied up at a time. In order to avoid injury to the nerves a roller bandage or any other soft pad should be placed under the tourniquet over the path of the larger vessels. The bandage should not remain in place for more than two hours, otherwise muscular paralysis or necrosis of the skin may result. As a rule, one-half hour is sufficient. The bandage is to be removed slowly for obvious reasons.

<sup>1</sup> Ztschr. f. exper. Pathol. u. Therapie, 1910, vii, 290.

<sup>2</sup> Beitr. z. Klinik d. Tuberkulose, 1913, xxviii, 1.

**Artificial Pneumothorax.**—In cases in which the above measures are of no avail, the induction of an artificial pneumothorax may be considered, provided it can be ascertained in which side of the chest the bleeding is going on. This point is discussed elsewhere in this book. But it should be stated that in very acute cases in which the exsanguination is sharp and brisk, there is usually nothing to lose and, even when we are not sure, we are justified in inducing a pneumothorax in the pleura of the lung which is most likely the source of the bleeding as shown by clinical indications.

**Medicinal Treatment.**—It will be noted that with exception of emetin we have left the drugs which have been used for the purpose of allaying pulmonary hemorrhage to the end. The reason is that we do not know of any drug which will stop hemorrhage in the lung. It seems to me that the reputation of some drugs as pulmonary hemostatics has been acquired on the basis of the fact that the vast majority of hemorrhages stop spontaneously; anything will do and receive the credit. This appears to be the consensus of opinion of phthisiotherapeutists at present, although no less an authority than Albert Robin<sup>1</sup> says that he feels constrained to protest vigorously against the allegation that medicinal agents are impotent, and are only given credit for their psychic effects. To be sure, he says, there are many cases of hemoptysis which stop spontaneously with or without treatment; there are others which cannot be controlled by any treatment. But between these two extreme types there are many cases in which medicinal treatment has a decidedly beneficial influence. Among these drugs Robin mentions ergot, calcium chlorid, gelatin, trinitrin, adrenalin, ipecac, digitalis, etc.

**The Nitrites.**—The nitrites have been found efficient in checking the bleeding from the lung. They are known to lower the blood-pressure and this may be the cause of their efficacy. Macht<sup>2</sup> found experimentally that the nitrites cause a constriction of the pulmonary vessels and at the same time they are efficient peripheral and splanchnic vasodilators. As usually given in 2 or 3 drops, amyl nitrite is often inefficient. I found that J. E. Squire's<sup>3</sup> suggestion to give 10 to 15 drops, dropped on a handkerchief which is placed before the patient's mouth and nose, is best. Immediately the face becomes red and congested and the hemorrhage stops. It may be repeated several times during the day. In more copious hemorrhages, where the nose gets blocked up with blood and clots, it may be necessary to put from 30 to 50 minims on a piece of lint and hold it over the patient's mouth. It may have to be repeated and the only complaint heard from the patient is that it produces a feeling of nausea. C. Fochi<sup>4</sup> says that when administered as soon as the first traces of blood-spitting are

<sup>1</sup> *Thérapeutique usuelle de la tuberculose*, Paris, 1912, p. 294.

<sup>2</sup> *Jour. Amer. Med. Assn.*, 1914, lxii, 524.

<sup>3</sup> *Clinical Journal*, 1909, xxxiv, 155.

<sup>4</sup> *Gazzetta degli Ospedali*, 1908, xxix, 114.

seen, copious hemorrhages may be prevented. But this is open to question. Fatal hemoptysis only rarely begins with streaky sputum. It is copious from the start, as a rule.

In slow bleeding, nitroglycerin, given in small and frequently repeated doses, as recommended by Flick, is often of service. When administered in 2- to 4-drop doses of the 1 per cent. alcoholic solution it produces the same effect as amyl nitrite, but slower and more lasting effects are observed. Tablets are not to be trusted because they are often inert, as has been shown by George B. Wallace and A. I. Ringer.<sup>1</sup> The 1 per cent. solution, as represented by the pharmacopœal spirits, is the best form in which glonoin should be administered. The following formula may be prescribed:

R—Spirit. glonoini . . . . .	ʒj	4.0
Aquæ aurantii flor. . . . .	ʒj	30.0
Aquæ destil. . . . . ad	ʒiv	120.09
M. S.—One teaspoonful three or four times a day.		

**Adrenalin.**—During recent years adrenalin has been used quite extensively for hemoptysis. It has been stated that it works well in cases where it is likely that the hemorrhage is due to the erosion of a medium-sized vessel, and that in acute inflammatory conditions of the lung it is contra-indicated. It increases the heart action and contracts the bloodvessels, especially of the intestines, kidneys, and spleen, and thus increases the blood-pressure. But Gerhardt says that the bloodvessels of the lung are but slightly contracted, while Frey found that in a bleeding lung in a rabbit the vessels dilated and the flow of blood was increased after the administration of adrenalin, and Macht<sup>2</sup> found experimentally that it causes a powerful constriction of the pulmonary artery. Moreover, according to von den Velden, the coagulability of the blood is increased 50 per cent. after the subcutaneous administration of the remedy. Clinical experience with this drug has not convinced the writer of its efficacy in hemoptysis and it has therefore been discarded.

**Ergot.**—Ergot has been given in large doses (a teaspoonful of the tincture every three or four hours; ergotin hypodermically). But it has been conclusively shown that it increases the pressure in the lesser circulation, just what we want to avoid. In the writer's experience it has never been of any value, often decidedly harmful. The same may be said about digitalis.

**Atropin.**—Atropin administered hypodermically, in doses of  $\frac{1}{80}$  grain every three or four hours, according to indications, has been of more service than ergot or digitalis. Still, in some cases the writer has observed an increase in the hemorrhage soon after its administration.

**Gelatin.**—With a view of increasing the coagulative power of the blood gelatin has been recommended by Dastre and Floresco.<sup>3</sup> Four

<sup>1</sup> Jour. Amer. Med. Assn., 1909, liii, 1629.

<sup>2</sup> Jour. of Pharmacol. and Exper. Therap., 1914, vi, 13.

<sup>3</sup> Compt. rend. de la Soc. de biolog., 1896, iii, 243.

to 6 ounces of a sterilized 3 per cent. solution of gelatin is injected under the skin of the abdomen or thigh. Great care must be taken in preparing the solution, as well as while injecting it because severe cases of sepsis, even tetanus, have been reported. Altogether it is not a harmless procedure—it is painful, leaves painful infiltrations at the site of the injection, often provokes fever, and is followed by urticarial eruption. If gelatin is used at all it should be given by mouth. The patient may be given jelly made from calves' legs, etc., or gelatin may be mixed with milk; or a concentrated solution may be administered per rectum.

**Calcium lactate, acetate, chloride, etc.,** are other time-honored remedies given with a view of increasing the coagulability of the blood in doses of 10 to 20 grams repeated four to six times a day. Their utility is doubtful; all that may be said about them is that they are painless and harmless.

**Blood Serum.**—The use of blood serum in hemophilia has suggested its application in hemoptysis with a view of increasing the coagulability of the blood. Horse serum may be used in doses of from 20 to 40 c.c. subcutaneously. Inasmuch as at present diphtheria antitoxin is everywhere available, it may be used. But manufacturing chemists now have on the market appropriate preparations. It should not be used at long intervals several times for fear of anaphylaxis. I have tried it several times and was not favorably impressed with it.

**Venesection.**—With a view of producing a rapid fall in the blood-pressure venesection has been used in desperate cases of pulmonary hemorrhage. In the days of indiscriminate bleeding, this was one of the standard therapeutic measures, but even at present many authors recommend it. Bonney recommends it when the blood-pressure is abnormally high even in small initial hemoptysis, and also in bronchopneumonia following pulmonary hemorrhage, when the right heart is dilated and there is pulmonary edema, cyanosis and coma. More recently A. G. Shortle<sup>1</sup> urged this method again in cases in which the bleeding is seriously interfering with the functions of respiration. "The prompt relief to the impaired respiration is not the only benefit rendered in such cases. The coughing and struggling for breath, with the coincident inspiring of blood and sputum into the air cells is also stopped, and the development of bronchopneumonia may be prevented." In persisting hemorrhages it is also indicated, according to Shortle; "it is safer to bleed from the arm than from the lung."

Of course this is rather heroic treatment, and involves great responsibility, especially when attending to patients in their homes. But in the desperate cases in which there is evidently nothing to lose, it may be given a trial when everything else has failed.

**Diet in Hemoptysis.**—In cases of slight hemoptysis with only streaky sputum, or when a few mouthfuls of blood are brought up, the diet

<sup>1</sup> Trans. Nat. Assn. Study and Prev. of Tuberc., 1915, xi, 147.

need not be changed. But in active and profuse hemorrhage all solid and hot food is to be interdicted. Inasmuch as the first indication is to reduce the blood-pressure, we must restrict the quantity of fluids ingested. Sudden or rapid filling of the bloodvessels with water increases the blood-pressure and may lead to an increase in the bleeding. In European resorts, where phthisis is treated with mineral waters, hemorrhagic cases have been excluded ostensibly for the reason that excessive ingestion of water induces hemorrhage. In very copious hemorrhages, fluids should be given only for the purpose of allaying thirst—a couple of ounces at a time. Swallowing small pieces of ice served this purpose best. Alcohol, coffee and tea, etc., should be discarded. Milk, eggs, scraped beef, etc., may be given in small quantities at a time.

Twenty-four hours after the cessation of the bleeding, irrespective of the clots expectorated with the sputum, we may begin to feed the patient guardedly. The general condition of the patient, as well as the concomitant symptoms should be our guides. A cup of milk every hour or two, cream, a raw egg, and some scraped beef may be given. On the third day ordinary feeding may be resumed, so that about five or six days after the hemorrhage a standard dietary is reached.

**Convalescence.**—During convalescence, even if there is no fever, or other complications, the patient is to be kept in bed for five or six days after the disappearance of all traces of blood from the expectoration. Resumption of exercises should be allowed gradually. It is best that for two or three weeks after even a moderate hemorrhage the patient should keep at comparative rest. The cough should be carefully controlled during that period and exposure, especially to intense sun rays, avoided. Patients who show a proclivity to frequently recurring hemorrhages are to be warned against all physical and mental excesses, and alcohol is to be strictly prohibited.

**Dyspnea.**—We have seen that subjective dyspnea is rare in many cases of chronic phthisis, and that the patients are only rarely short-winded, if at all. In some cases this symptom demands treatment.

Toxic dyspnea, due to progressive disease of the lung, is best treated by rest. It is always accompanied by fever and the treatment directed to remove the pyrexia usually helps along in the direction of relieving the air hunger. During acute exacerbations in the course of chronic phthisis, toxic dyspnea is very frequent and the treatment is clearly defined.

Dyspnea is often due to some preëxisting disease. This is the case with pulmonary emphysema, asthma, cardiac and renal disease. The treatment is that of the underlying pathological condition. In those having emphysema or asthma, the iodides are very often of immense help, provided there is no tendency to hemoptysis. For the nocturnal attacks of dyspnea, morphin or heroin may have to be given.

Dyspnea may be due to some acute or subacute complication, such as pleurisy, with or without effusion, spontaneous pneumothorax,

etc. The treatment is considered in the sections dealing with these complications. In the terminal stages of the disease the air hunger may only be relieved by large doses of morphin or heroin and no patient should be denied these solacing remedies. The dangers of habit formation should not be thought of at this stage of the disease.

**Cardiac Weakness.**—Patients who suffer from tachycardia or cardiac palpitation, permanent, or provoked by mild exertion or excitement, must be kept at perfect rest in bed. Smoking and the consumption of alcohol and coffee are to be interdicted and all forms of nervous and emotional excitement are to be avoided. At times these cardiac disturbances are due to gastric derangement and may call for modifications in the quantity and quality of the food.

In many cases, especially in the advanced stages, palpitation is due to cardiac displacement, especially in left-sided lesions in which the heart is drawn upward and to the left. Rest is the only remedy we have for this condition.

From whatever cause cardiac weakness arises, it may at times become acute; collapse is not uncommon after some excitement or overexertion. Now and then a patient dies suddenly as a result of heart failure. For collapse, hot drinks of whisky, warm applications to the extremities, and some stimulants like camphor, strychnin, etc., are to be administered hypodermically.

In the far-advanced stages there is acute dyspnea, cyanosis, and edema owing to cardiac failure resulting from the extensive lesion, toxemia, etc. These terminal symptoms are treated with digitalis, though in my experience this drug has very little influence on the heart at this stage. In most cases the subjective feeling of weakness and air hunger are best relieved by liberal doses of morphin, or heroin.

**Insomnia.**—In phthical patients insomnia may be due to various causes and it is not advisable to resort to soporific medication in every case. Rest and fresh air in the sleeping room may induce sleep; so may avoidance of a heavy meal late in the evening, a warm bath before retiring, etc. These means will suffice in most of incipient cases in which the sleeplessness is due to worry on account of the seriousness of the ailment.

In incipient cases insomnia may be due to the cough which keeps the patient awake, and the indications are those discussed when speaking of the treatment of cough. When due to digestive disturbances, it is to be treated accordingly. In the advanced stages it is often due to the fact that the patient is lying at perfect rest during the whole day, and sleeps several hours for an hour or so at a time. The patient is then to be kept awake during the day. In some cases hypnotic drugs must be given, and of these sulfonal or trional, in 10- to 15-grain doses, may be administered; 3 to 6 grains of veronal will serve the purpose in some cases. If the treatment has to be prolonged, the drugs may have to be alternated. In the far-advanced stages only large doses of morphin may give relief.

**Pains in the Chest.**—Most of the pains in the chest complained of by tuberculous patients may be relieved by the administration of some placebo or the application of a mustard plaster, dry cupping, tincture of iodine, etc. In some cases it is necessary to administer some of the coal-tar analgesics or salicylates. Small doses of antipyrin, phenacetin, pyramidon, etc., with caffeine may be given. Sodium salicylate or aspirin gives relief in many cases. But on rare occasions we meet with patients in whom the pains in the chest are so severe as to require the administration of a dose of codein or morphin. When due to intercurrent pleurisy, strapping of the chest with adhesive plaster is indicated. The pains in the shoulder, which are very acutely felt especially during the night, are very difficult to manage. The coal-tar analgesics and the salicylates usually give no relief, and often even safe doses of morphin fail. Hot applications to the affected part, or rarely the actual cautery, may be necessary.

**Anorexia.**—Many patients have a good appetite; even when the fever is comparatively high the desire for food may be retained, which is not observed in other febrile diseases. But in others it is defective or inadequate to induce them to ingest a sufficient quantity of food for the replenishment of the inroads on their bodies made by the disease. It has been my experience that their number is not very large among those who are well instructed along the line of proper food and nourishment.

Medicinal treatment is not the first thing to give in anorexia. Outdoor life, regulated exercises, regularity of meals, etc., suffice in most cases to improve the appetite to the desired degree. In many it will be found that dietetic errors are at the bottom. The traditional and stereotyped advice "plenty of milk and eggs" given indiscriminately, is more responsible for disgust for food than any other single factor. Drinking two or even three quarts of milk a day, and swallowing six to twelve raw or soft-boiled eggs, overload and often dilate the stomach, produce congestion of the liver, and create a disgust for all kinds of food. While some patients, who may be considered dietetic curiosities, may keep up with such a regime for weeks and even gain in weight, in the vast majority the digestive organs revolt, the palate loses its taste for food altogether and, coupled with diarrhea or constipation, the functions of assimilation fail.

In this class of patients we may note with satisfaction a remarkable change soon after the quantity of milk and eggs is reduced, or they are altogether discarded for a time. We must never neglect to tell our patients that as long as the appetite and digestion are good, they need not make any changes in their accustomed diet, excepting perhaps to increase the quantity, which is very desirable. With a variety of food-stuffs it is usually easy to consume more than before the onset of the disease. Instructions along the lines of good cooking should never be neglected. Among the poor and moderately well-to-do it has been my habit to send for the mother, wife, or sister of the patient and urge her



to exercise special care in the preparation of the food and to cater to the palate of the patient. The person who has prepared food for the patient for a long time knows best what he will relish. Of course the teeth are to be examined and repaired in case caries are found, and proper instructions as to mastication are to be given.

In most cases the appetite can be improved by corrections of any of the just mentioned errors without any medication at all. All are to be told in plain language that their only chance for recovery lies in consuming proper food and plenty of it; that they can best be cured through their stomach, and that they must eat even if the desire for food is not at its best. This often has the desired effect. When the patient finds that with proper food he gains in weight he is encouraged to eat more. The gain in weight is usually seen best during the first month or two, but after a considerable increase the gain slackens. As long as he holds his own at his former weight, or little above, there is nothing to worry about.

**Gastric Disturbances.**—In some cases we must resort to medication to provoke an appetite. I consider creosote as the drug which acts the best. Small or moderate doses of creosote or any of its derivatives—creosote carbonate, guaiacol, guaiacol carbonate, etc.—may be given and the appetite and digestion promptly improve. In others we may give bitter tonics—the tinctures of *nux vomica*, *condurango*, *cinchona*, etc. *Orexin tannate* is also good in 5-grain doses in powder or tablet form taken half an hour before meals. When there is diarrhea, this drug is very good. I have used the following with good results:

R—Tinet. nucis vomicae . . . . .	ʒij	8.0
Acid. nitrohydrochlorici dilut. . . . .	ʒiij	12.0
Tinet. gentianae comp. . . . .	ʒij	64.0
Tinet. cardamomi comp. . . . .	q. s. ad ʒiv	120.0
M. S.—One teaspoonful well diluted in water three times a day before meals.		

The *nux vomica* may be replaced by *condurango* and the nitrohydrochloric acid omitted in cases in which they are contra-indicated. In obstinate cases stomachic medicaments are to be changed often.

In hyperacidity dietetic changes are to be made according to indications, and it is always to be borne in mind that it may be due to overfeeding. Often medication is necessary. I have had good results with the following:

R—Magnesii oxidi . . . . .	ʒiv	16.0
Sodii bicarbonatis . . . . .	ʒj	32.0
Extracti belladonnae . . . . .	gr. ij	0.13
M. ft. chart. No. xxiv div.		
S.—One powder three times a day after meals.		

Or the following effervescent powder may be given: 30 grains of bicarbonate of sodium in one powder, and 10 grains of tartaric acid in another. Each of these is to be dissolved in half a tumbler of water, then added one to the other and swallowed during effervescence.

Some are relieved by a tablet of  $\frac{1}{100}$  grain of atropin sulphate given after meals.

**Constipation.**—Constipation is another of the troubles of the phthisical which often interferes with the favorable progress of the case. It is best combated by proper dietetic measures, especially increasing the quantity of fruits and vegetables, fresh and cooked. But mildly laxative drugs must be given in many cases. Before giving them we must make sure that it is not one of the anodyne drugs, codein, morphin, dionin, etc., which is responsible. Phenophthalein appears to be the best, and 3 to 5 grains may be given, and next to it cascara sagrada in appropriate doses.

In the advanced stages, when diarrhea is apt to alternate with constipation, laxative drugs are to be used with caution. They may induce an uncontrollable diarrhea. It is always better to first try proper changes in the diet, or the effects of some special food. Thus, I find that buttermilk will cause a movement of the bowels better than any medication in some tuberculous patients.

**Diarrhea.**—We have seen that diarrhea in the tuberculous is not always due to ulcerations in the intestines and that the latter may exist, while the patient is constipated. In many cases the diarrhea is due to chronic catarrh of the bowels induced by swallowed sputum and the patient is to be warned against this very bad habit. In others it is due to consumption of large quantities of raw milk, and this must be corrected.

In case the diarrhea is due to tuberculous ulceration or amyloid degeneration of the intestines, it is often very difficult to manage. The patient must remain in bed and appropriate changes be made in the diet. Fluids in general are to be reduced in quantity, especially cold drinks. The great majority of vegetables, salads, fruits—raw or cooked—pastries, rye bread, fats and sweets are to be avoided. While most patients tolerate milk very well, there are many who do not and, in obstinate cases, it is advisable to discard it for a few days and watch the effects. Bouillon and soups should be given without the addition of vegetables; eggs, butter, scraped or finely minced beef, boiled fish, and oysters may be allowed, but no lobster. Of the vegetables allowed the following may be mentioned: Rice, sago, etc., boiled in milk or served with cream, mashed potatoes, etc.

In many cases medicinal treatment must be given to control the frequent stools. The ancient "styptic" remedies, such as lead acetate, iron, alum, etc., are worthless in the vast majority of cases. But the modern preparations of tannin, such as tannigen, tanalbin, etc., are occasionally of service in large doses, and should be given a trial. The subnitrate of bismuth should be given in doses of 10 to 15 grains five or six times a day. But in most cases opium must be used, more or less. Bismuth or tannigen may be given in powders combined with fairly large doses of Dover's powder, or the official tincture of opium in 5- to 10-minim doses three or four times a day.

R—Tannigeni . . . . .	ʒiij	12.0
Bismuthi subnitrat̄is . . . . .	ʒvj	24.0
Resorc̄inolis . . . . .	gr. ix	0.6

M. ft. cachet No. xviii.

S.—One cachet four times a day.

R—Bismuthi subnitrat̄is . . . . .	ʒj	32.0
Tinct. opii deodorati . . . . .	ʒij	8.0
Aquæ cinnamoni . . . . .	q.s. ad ʒiv	120.0

M. S.—One teaspoonful four times a day.

When bismuth subnitrate fails we may try the subgallate in 10- or 15-grain doses with or without opium. There are, however, many cases in which everything, even the administration of heroic doses of opium, fails to stop the diarrhea and we must be content with relieving the pains.

Some of these patients complain of tenderness or pain in the abdomen. This is best relieved by hot fomentations. In the later stages, when emaciation is extreme, the extremities are to be kept warm and the unfortunate patient should not be denied the merciful relief of morphin in large doses.

## CHAPTER XXXIX.

### OPERATIVE TREATMENT—ARTIFICIAL PNEUMOTHORAX.

**Historical Note.**—Spontaneous pneumothorax has been the most dreaded of complications of phthisis and experience has taught that the vast majority of patients who suffer from this accident succumb. But some have observed that a pneumothorax may be what the French call “providential” and exert a rather salutary influence on the symptoms of the underlying disease. In fact as far back as 1822 James Carson,<sup>1</sup> a physiologist at Liverpool, suggested the advisability of artificially inducing pneumothorax in phthisis for therapeutic purposes and performed some animal experiments with a view of working out a suitable technic. In his book on diseases of the chest, published in 1837, that acute clinical observer, William Stokes,<sup>2</sup> has this to say: “The proper symptoms of phthisis are in many cases arrested, and singularly modified, by the occurrence of the new disease (pneumothorax). I have often found that after the first violent symptoms had subsided, the hectic ceased, the phthisical expression disappeared, the flesh and strength returned; and in this way the patient has enjoyed many months of comfortable existence, and was only disturbed by dyspnea and the sound of fluctuation on exercise.” In his book on *Diseases of the Lungs*, published in 1860, Walter Hayle Walshe<sup>3</sup> says: “In some recorded cases of actively advancing phthisis, the first sufferings of accidental perforation having passed, it has certainly appeared, though the signs of hydropneumothorax remained, that the phthisical symptoms themselves underwent improvement. But an occurrence so rare gives no warranty for the fanciful proposal to treat phthisis by producing artificial pneumothorax.” This shows clearly that the method was suggested in England long before Forlanini has done it in Italy. During the course of the nineteenth century many other physicians have reported experiences similar to those of Stokes and Walshe just quoted.

It was, however, C. Forlanini,<sup>4</sup> of Pavia, who first induced a pneumothorax for therapeutic purposes and reported his experiences in 1894. Independently of Forlanini, John B. Murphy,<sup>5</sup> of Chicago, did the

<sup>1</sup> The Elasticity of the Lungs, Trans. Roy. Soc. London, 1820; Essays, Physiological and Practical, Liverpool, 1822.

<sup>2</sup> A Treatise on Dis. of the Chest, New Sydenham edition, p. 455.

<sup>3</sup> A Practical Treatise on the Diseases of the Lungs, American edition, Philadelphia, 1860, p. 250.

<sup>4</sup> Gazz. d. osped., 1882, iii, 537, 585, 601, etc.; Gazz. med. di Torino, 1894, lxxv, 381, 401.

<sup>5</sup> Jour. Amer. Med. Assn., 1898, xxi, 151, 208, 281, 341.

same in 1898. But for some time no notice was paid to this method of treatment until Brauer, Spengler, and some others took it up in Germany. At present it is one of the recognized methods of treatment of certain cases of pulmonary tuberculosis. That it is a valuable method will be appreciated when it is borne in mind that it is mostly indicated in cases in which everything else has been tried and found wanting; in other words, when there is everything to gain and nothing to lose. Contrasted with other methods of treatment, which are nearly always stated to exercise their alleged curative effects only during the incipient stage of the disease, when diagnosis is often doubtful and spontaneous cures not uncommon, it is to be considered one of the best therapeutic procedures we have at present for the cure of phthisis.

**Principles Underlying the Treatment.**—The aim is to introduce into the pleural cavity a sterile and harmless material which will collapse the lung on the affected or more affected side of the chest. The lung is thus put at rest and given an opportunity to heal. We have already seen that functional rest is as important in phthisis as in other diseases. In surgical tuberculosis rest has been more effective as a curative agent than all other methods. Rest has also been used with beneficial results in other diseases, notably general rest in functional nervous diseases as was worked out by Weir Mitchell; tracheotomy in certain laryngeal obstructions, gastro-enterostomy in cancer and especially ulcer of the stomach, enterostomy in certain diseases of the lower bowels and rectum, etc.

The lung is one of the organs of the body which never rests but expands and contracts at least 12,000 times per day throughout life. With an artificial pneumothorax we can place one lung at rest almost as effectively as the splint puts at rest a tuberculous joint, without endangering the life of the patient. Moreover, the lung is the only organ in the body which is constantly in a state of distention. Even after the most forced expiration it does not collapse utterly. Any solution in continuity in the pulmonary tissues remains separated and there appears to be no tendency to bring about the union of the diseased parts or to facilitate the process of healing by coaptation. Inflating gas into the pleural cavity and collapsing the lung we achieve two objects: The lung is immobilized at its root, and it is compressed by the gas in the pleural cavity and the retraction of its elastic tissues. Its volume is greatly reduced, diseased parts and walls of cavities are brought into apposition, so that they may cicatrize by the formation of connective tissue.

Pneumothorax does even more than afford rest to the diseased lung. By compression it empties the lung of its contents. The pus and cheesy detritus in cavities, the inflammatory exudates in the alveoli and bronchioles are all squeezed out as from a sponge, removing the main source of toxic absorption. It also limits the diseased focus and prevents its spread, so that the healthy parts of the lung remain so while

the lesion is in time converted into a cicatrix, or is encapsulated. As a result of drainage, mixed infection is eliminated and prevented. The fact that the air current entering through the trachea cannot circulate within the collapsed lung tissues, prevents superinfection of healthy parts of the organ with emboli of detritus carried from one part to another along the bronchial tree and mixed infection with microorganisms other than tubercle bacilli, which may be brought in with the air current, is avoided.

The circulation of the blood is impeded in the collapsed lung, but there occurs a venous or passive hyperemia which is known as an important factor in the defence of tissues against tubercle bacilli. The comparative protection against tuberculosis enjoyed by cardiacs is ascribed to the venous hyperemia of the lungs. The lymph channels of the collapsed lung are compressed, as has been shown by Shingu,<sup>1</sup> who subjected animals with induced pneumothorax to the inhalation of soot, and at the autopsy found that the collapsed lung remained free from soot. Animals were compelled to inhale large quantities of soot, and subsequently pneumothorax was induced, and when they were finally killed it was found that the free lung was darker than the collapsed lung. This tends to show that the circulation of lymph, which is the main factor in removing inhaled particles from the lung, is impeded or arrested because of stasis of lymph in the compressed lung. In this manner the absorption of toxins from the lesions into the general circulation, is impeded or arrested in pneumothorax, the clinical phenomena of phthisis, such as fever, nightsweats, weakness, etc., are prevented, and the body is thus given an opportunity to recuperate. Moreover, the lymph stream being unable to carry away bacilli from the lesion, the process is localized to the affected areas. These points have been found clinically and at the autopsy table, and experimentally by Forlanini,<sup>2</sup> Brauer,<sup>3</sup> Saugman, Graetz,<sup>4</sup> Robinson and Floyd,<sup>5</sup> Hamman and Sloan,<sup>6</sup> G. M. Balboni,<sup>7</sup> Henry Schwatt, Ralph C. Matson, A. G. Shortle, and many others.

**Technic.**—The technic of the induction of a pneumothorax is simple but not devoid of danger and even fatal accident. The object is to inject gas into the pleural cavity, and not anywhere else. Forlanini developed a technic which is both painless and bloodless. Murphy, without knowledge of Forlanini's work, developed a practically similar technic. Brauer was not satisfied that the Forlanini-Murphy method is safe and advocated the open incision method.

**The Brauer Method.**—This consists in incising the chest wall, dissecting down to the pleura by cutting through the fascia, and separating

<sup>1</sup> Beitr. z. Klinik d. Tuberkulose, 1908, xi, 1.

<sup>2</sup> Ergebn. d. inner. Med. u. Kinderheilk., 1912, ix, 621.

<sup>3</sup> Beitr. z. Klin. d. Tuberkul., 1909, xii, 49; xiv, 419; 1911, xix, 1.

<sup>4</sup> Ibid., 1908, x, 249.

<sup>5</sup> Arch. Int. Med., 1912, ix, 452.

<sup>6</sup> Johns Hopkins Hosp. Bull., 1913, xxiv, 264.

<sup>7</sup> Boston Med. and Surg. Jour., 1914, clxxi, 697, 955.

the intercostal muscles with a blunt instrument in the direction of their fibers. When the parietal pleura is exposed, it is punctured with a blunt needle or cannula and the gas is allowed to flow in by aspiration of the pleural cavity or by pressure when indicated. This method has failed to get many adherents for many reasons. But few patients want to submit to a cutting operation. Then there is an obvious danger of sepsis which may, of course, be avoided by the usual methods. I have found no reason for resorting to the bloody operation and feel confident that if this was the only available method

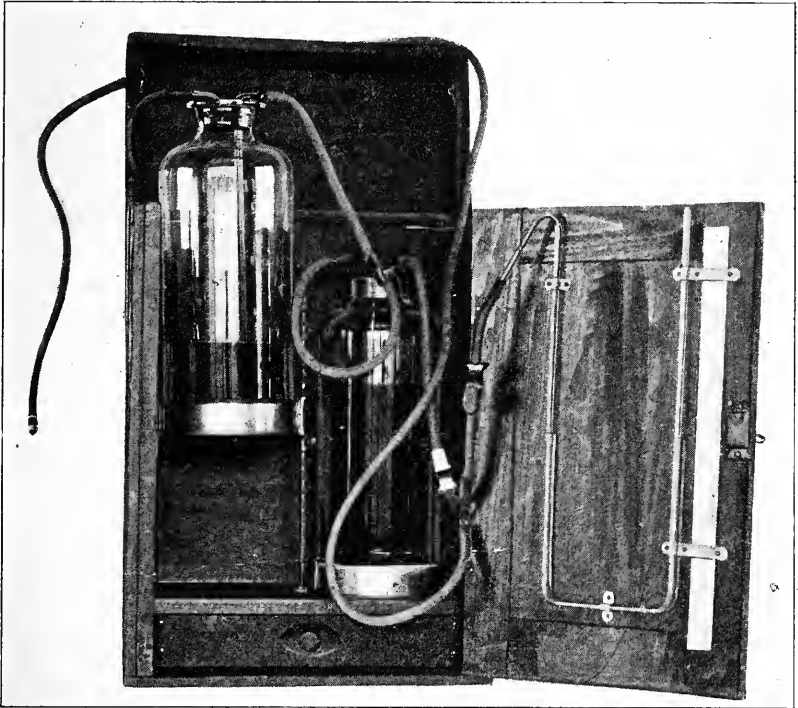


FIG. 83.—Robinson's modification of the Brauer apparatus for inducing pneumothorax.

of inducing an artificial pneumothorax we should find very few patients willing to submit.

Very few now practise this open incision method and most of those who do it make use of it only occasionally when the Forlanini method fails because of pleural adhesions.

**The Forlanini-Murphy Method.**—It consists in a simple, bloodless puncture of the chest wall with an especially constructed hollow needle which is connected with a gas reservoir and a water manometer through a T-shaped tube. When the lumen of the needle punctures the costal pleura the gas is allowed to flow into the pleural cavity by

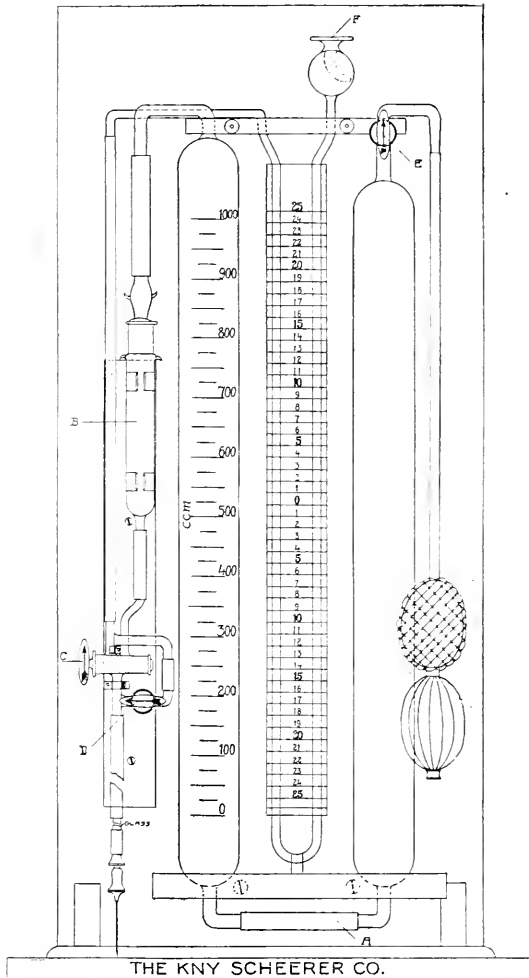


FIG. 84.—Forlanini-Saugman-Murali apparatus for the induction of pneumothorax. This apparatus consists in the main of two glass tubes, twenty-four and a half inches high and about two inches in diameter and a U-shaped manometer tube, the latter filled with an alcoholic solution of methylene-blue and mounted in the centre of the board in front of a graduated porcelain scale. The two large tubes are joined by means of rubber tubing under the base *A*. The tube to the left is graduated to 1000 c.c. and the other is plain. They are filled with water up to 500 c.c.

The graduated tube to the left is filled from the tank with the gas to be introduced into the pleural cavity, and the gas displaces the water which rises correspondingly in the large plain tube to the right.

When filling the apparatus with gas, the rubber tubing from the tank is to be connected with a rubber gas-bag to the opening below the stopcock *C*.

Stopcock *D* should stand vertically.

Stopcock *C* should be turned so as to connect through the filter and into the graduated cylinder.

Stopcock *E* on the top of the non-graduated tube should be turned so as to allow the air in this tube to escape when the gas forces the water into it. When the graduated cylinder is full of gas, stopcock *C* should be closed.



the suction or negative pressure in that cavity, as well as by some positive pressure which must at times be used at the gas reservoir.

Simple as this operation appears to be, there are certain difficulties to be overcome and dangers to be avoided. The main difficulty is to pass the needle as far as the costal pleura, puncture it, and avoid penetrating the visceral pleura and the lung. The dangers are mainly in allowing the gas to flow into places other than the pleural cavity, especially into a bloodvessel thus causing gas embolism which, while not invariably fatal, yet is sufficiently menacing to be dreaded by all who are doing this sort of operation.

**Apparatus.**—To avoid this accident various forms of apparatus have been invented. As is usual, they are all based on one main principle—the manometer which was introduced by Saugman. Each apparatus consists primarily of two graduated bottles connected by tubing, one containing the gas to be injected and the other some fluid, so that the fluid flows from its container into the other bottle, displacing the gas which is sucked or pressed into the pleural cavity through a tube and an especially constructed needle. This last mentioned tube is T-shaped or provided with a three-way stopcock, of which one limb communicates with the gas bottle, the second with the needle, and the third with the manometer. At any moment during the operation we can open or close the tube leading to the manometer or the gas reservoir.

As has been said, all the instruments for the induction of a pneumothorax are constructed on this simple principle, but it is amazing how some have succeeded in complicating it by adding various attachments which make them unwieldy and easily disordered. The experience that a machine in order to be successful must be of the simplest construction consistent with efficiency, holds good here. I have been using Forlanini's apparatus as modified by Saugman<sup>1</sup> and von Muralt,<sup>2</sup> (Fig. 84) and also the Robinson apparatus (Fig. 83).

**The Function of the Manometer.**—The entire safety of the apparatus lies in the manometer which has been called by Edward von Adelung<sup>3</sup>

<sup>1</sup> Beitr. z. Klinik d. Tuberkulose, 1914, xxxi, 571.

<sup>2</sup> Ibid., 1910, xviii, 359.

<sup>3</sup> Jour. Amer. Med. Assn., 1914, xlii, 1914.

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Funnel *F* connected with the manometer tube serves for the filling of the manometer tube to zero with an alcoholic solution of methylene-blue.

The graduated glass tube is connected with the glass tube *B* which is filled with sterilized gauze and serves as a filter. The three-way stopcock *C* connects with the manometer as well as the gas cylinder, thus showing the oscillations when the needle is in the pleural cavity. When stopcock *D* is turned horizontally it permits the manometric reading showing the degree of oscillation while the gas is still flowing.

After the needle has been properly inserted into the pleural cavity and stopcock *C* turned to the graduated tube, the gas will be forced out by the weight of water which is contained in the plain tube. When extra pressure is required, a small rubber tube is connected with the plain tube, so that the remaining water may be gently forced into the graduated tube.

The manometric scale is divided into 50 centimeters, 25 above and 25 below zero, indicating respectively negative and positive pressure.

the heart of the apparatus. While the needle passes through the skin, subcutaneous tissue, muscles, and fascia before the piercing the costal pleura, the manometer records atmospheric pressure, but as soon as it enters the pleural cavity the air in the connecting tube becomes rarefied because the vacuum in the pleural cavity aspirates its air content, and the fluid in the closed limb of the manometer is sucked up toward the needle, *i. e.*, from the open into the closed limb and a distinct difference in the levels of the fluid is evident. Moreover, when the lumen of the needle is really in the pleural cavity the respiratory movements of the lung are recorded in the manometer which shows distinct oscillations of the levels of its fluid.

This explanation of the work of the manometer, which is found in most works on the subject, is unsatisfactory. The fact is that normally there is no pleural cavity at all because the parietal and visceral pleura lie tightly, one on another; nor can we speak of negative pressure between the two pleural sheets because the word "pressure" is here used

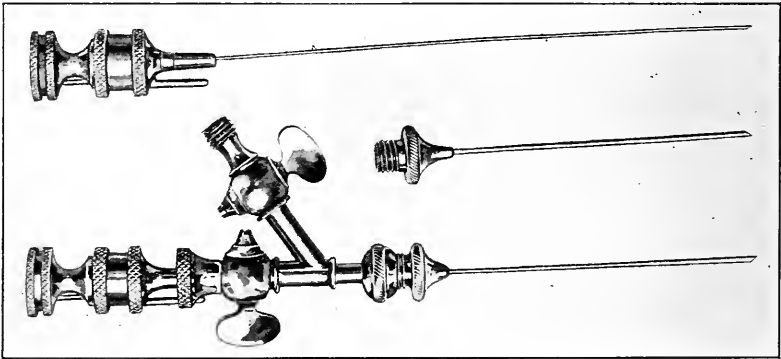


FIG. 85.—Brauer-Floyd-Robinson needle.

in the sense of gas pressure which can be measured with a manometer; but such a negative pressure does not exist between the two pleural sheets. The manometric readings when the lumen of the needle is in the pleura are better explained by Brauer, Piéry,<sup>1</sup> and Moritz<sup>2</sup> in the following fashion: The lung must be considered as an organ fixed at its root and kept in a state of equilibrium by the pressure of the atmospheric air within the air passages and by the elastic tension of its tissues. There is a constant tension of the lung from the roots to the periphery at the thoracic walls. The force of this traction is equal to the absolute elastic tension in the given direction, minus the atmospheric pressure which prevails within the air passages and so prevents its collapse or retraction from the periphery to the hilus. The intrapleural pressure, therefore, never differs much from the atmospheric pressure, as

<sup>1</sup> La pratique du pneumothorax artificiel en phtisiothérapie, Paris, 1912.

<sup>2</sup> Münch. med. Wehnschr., 1914, lxi, 1321.

has been shown by W. Parry Morgan,<sup>1</sup> and in consequence any gas drawn into the cavity will not be appreciably rarefied. The volume of gas which will have passed from the connecting tube into the pleural cavity will be practically equal to the amount of fluid which will have passed from the open to the closed limb of the manometer. This volume would, when the negative pressure stands at 15 cm. of fluid in a manometer tube of 0.3 cm. bore, measure less than 1 c.c.

This is enough to separate the sheets of the pleura, if there are no adhesions. But owing to the elastic tension of the lung and the atmospheric pressure within the air passages, there is actually shown a negative pressure in the manometer. A little reflection will explain why this negative pressure will be stronger during inspiration because of the greater distance at that period between the root and the periphery, and less during expiration. With the increase in the quantity of gas introduced into the pleural cavity the tension of the lung will obviously decrease and with it the negative pressure, until finally a point is reached when the pressure in the gas-containing pleural cavity is 0 and later even becomes positive.

Bearing in mind these simple principles of the manometer, we are in a position to guard against the most important of the accidents which are liable to happen during the operation. In patients with pleural cavities free from adhesions, ordinary and careful attention to the manometer will suffice to guard against mishaps. The manometer shows conclusively whether the lumen of the needle is in the pleural cavity or not. It also gives reliable information as to the state of the pleural cavity with particular reference to adhesions, showing whether they are dense and extensive, or of slight extent and may be separated and broken up by an increase in the intrapleural pressure with the gas. During the course of the treatment we are able to ascertain, with the aid of the manometer, whether the nitrogen has been absorbed and a refill is necessary; whether the lung has been completely immobilized or has remained expansile. When it is found that the intrapleural pressure increases, and this cannot be attributed to excessive gas insufflations, it indicates pleural effusion. The difficulties in cases with pleural adhesions will be discussed later on.

**The Gas Used for Inflation.**—Because it was supposed that when oxygen is injected into the pleural cavity it is quickly absorbed, and

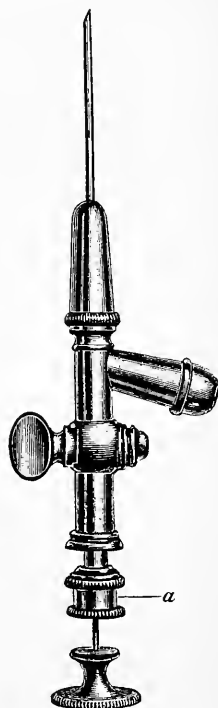


FIG. 86.—Saugman needle.

<sup>1</sup> Lancet, 1914, ii, 90.

that nitrogen will remain within that cavity for a longer time, this element was selected and most operators use it. But further experience has shown that atmospheric air is just as good. Webb, Gilbert, James and Haven,<sup>1</sup> and Tobiesen<sup>2</sup> have shown clinically and experimentally that nitrogen has little, if any, advantage over atmospheric air, because in either case diffusion of gases occurs so rapidly that within a few hours the proportion of the two gases, nitrogen and oxygen, is about the same. For this reason there is no necessity for using nitrogen. Air does just as well. Nitrogen is rather expensive when bought in tanks from manufacturers, and while most of the apparatus for the production of pneumothorax is portable, the large iron tank of nitrogen is not easily transported, and atmospheric air is to be given preference in private practice.

**The Selection of the Point for Injection.**—The first inflation must be carefully done and it is important to select a point to introduce the needle where no adhesions are likely to be encountered. Bearing in mind the anatomy of the chest and its viscera it is evident that the ideal point is along the anterior or posterior axillary lines, especially at the ninth intercostal space posteriorly for apical lesions, or in the third intercostal space just outside the mammillary line for lesions of the lower lobes. Of course, when we are free to choose, areas covered with thick muscles or the thick mammary gland in women are to be avoided. But we are not always free to choose, and any point must serve our purpose when the elective places are not available because of adhesions. It must also be emphasized that it is very difficult, often impossible, to avoid pleural adhesions with all the means of diagnosis at present at our command.

We are generally guided by the following principles: The chest is punctured as far as possible away from the main pulmonary lesion because pleural adhesions are most likely to be encountered over the diseased lung and, what is more important, while puncturing the lung is ordinarily harmless, in such places the needle may, however, penetrate cavities and produce a pyothorax. But adhesions are found everywhere and often where we least expect them. Physical diagnosis is apt to prove misleading and the fluoroscope and skiagraphy just as often may fail to reveal them. I have met with cases in which the skiagraph showed all the conventional signs of pleural adhesions but puncture revealed a free pleura and complete collapse was easily obtained with three or four inflations. More often yet the skiagraph shows a clear picture and it is concluded that the pleura is free, but puncture shows conclusively that there are adhesions.

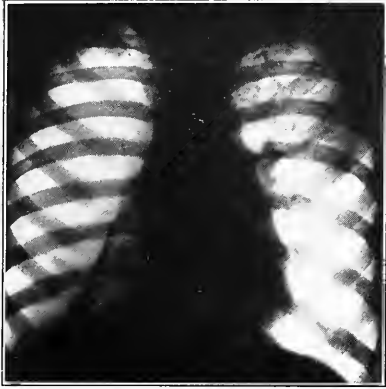
Forlanini is guided by tidal percussion of the margin of the lung, especially at the base. When he finds that the base line in the axilla shifts between 10 and 12 cm. during extreme inspiration, as compared with extreme expiration, he is convinced that the pleura is free. Good

<sup>1</sup> Arch. Int. Med., 1914, xiv, 883.

<sup>2</sup> Brauer's Beiträge, 1911, xxi, 109.

## PLATE XVII

FIG. 1



Complete pneumothorax in right pleural cavity, but there are several bands of adhesions running from the mediastinum to the diaphragm. Left lung shows moderate peribronchial infiltrations and a few calcified glands at the hilus. Lower two-thirds markedly emphysematous.

FIG. 2



Spontaneous pneumothorax following first inflation in an attempt at creating an artificial pneumothorax in left pleura. Diffuse peribronchial infiltration throughout right lung. Heart dropped, slightly displaced to the right. Pleuropericardial adhesions on left side.

FIG. 3

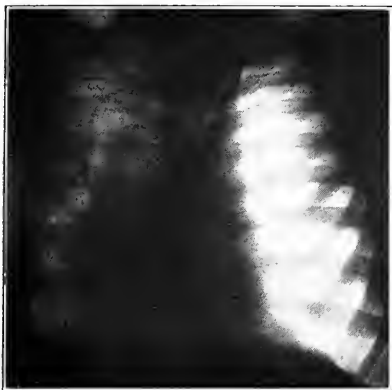


Incomplete pneumothorax in upper part of the right pleura. Owing to dense adhesions no more gas could be injected and the treatment was discontinued. Note the stomach at the left diaphragm.



## PLATE XVIII

FIG. 1



Complete pneumothorax of the left pleura. The right lung shows diminished aeration owing to fine, nodular infiltration and also to engorgement. Mediastinum completely displaced to the right.

FIG. 2



Complete pneumothorax of the left pleura with displacement of the heart to the right.

FIG 3



Darkness of right lung due to intense congestion after induction of a pneumothorax, excepting at the hilus, where it is due to enlarged glands and peribronchial infiltrations. One-half of the left pleura if filled with air, but the collapse of the lung was not effective in compressing a cavity with thick walls, situated in the first and second interspaces. Mediastinum displaced to the right.

FIG. 4



Pneumothorax localized in upper and lower portions of left lung, but separated by pleural adhesions at about the fourth rib, where also a cavity with dense walls is seen. These adhesions have interfered with the success of the pneumothorax.





mobility of the lung margins is the most important sign of freedom from pleural adhesions, according to Forlanini, but he adds that immobility is not a sure sign of such adhesions, and of obliteration of the pleural cavity. There are cases of extensive hepatization of the lung in which the mobility of the lung margin is defective or absent, yet the pleural cavity is free. Robinson and Floyd also consider percussion the most reliable guide and they say that the area presenting a note nearest approaching the normal resonance is most likely to be free of adhesions, while von Adelung seeks an area which is resonant and yields breath sounds.

It appears that the most reliable means of ascertaining whether or not the pleura is free is the attempt to enter it with the needle connected with a manometer. In case the first puncture does not yield negative pressure in the manometer—a very frequent occurrence, so that when one enters successfully with the first puncture he considers himself lucky—another attempt is made at a different point. I have made in one case four punctures before succeeding in entering the pleural cavity and in another twelve before giving up the case as not suitable for the treatment. Forlanini made fifteen punctures in one case before he finally succeeded.

The skin at the site selected for puncture is painted with tincture of iodine and the excess is washed away with alcohol. It is then frozen with ethyl chloride and an injection of one-third of a grain of novocain or cocain in 1 to 2000 adrenalin solution is made. At first the skin is infiltrated, then a few drops are injected into the intercostal muscles, and finally into the pleura. The latter must not be neglected; it appears to be the only known way of preventing pleural shock, of which we shall speak later on.

**Thoracocentesis.**—The patient is always in the recumbent position during the operation; either on an operating table or, preferably, in his bed. With a view of widening the intercostal spaces, the hand of the side to be operated upon is placed over the head. The selected intercostal space is carefully palpated with the index and middle fingers of the left hand to make sure of avoiding a rib when thrusting the needle into the chest wall. If a blunt needle is used the skin is first punctured with a tenotome. The needle is inserted and pushed slowly forward, passing through the subcutaneous tissue, fascia, and muscles. While the latter are passed the needle goes smoothly but when the endothoracic fascia is reached a certain amount of resistance is encountered, which is characteristic to the experienced hand. Often a snapping sound is audible. A similar, but stronger, resistance is felt when the pleura is passed and it is often difficult to decide with confidence as to whether it was the fascia or pleura which was punctured. "Never move the needle sidewise, for if it should be in the lung the latter may be easily torn by it" (Balboni). The manometer is the only means at our command to make sure of where the lumen of the needle is.

How far the needle is to be pushed depends on the thickness of the chest wall of the given patient. All efforts are to be made to avoid penetrating the lung. While in the vast majority of cases this is entirely harmless, on rare occasions it may prove a serious and even a fatal accident. We may induce a spontaneous pneumothorax, an accident which occurs more often that is generally appreciated.

The usual length of the needle, Floyd's modification of Brauer's, is 5 to 6 cm. This is excessive and Saugman's needle, which is only 3 cm. long, is at present used by me exclusively. Saugman noted in 100 cases in which he succeeded in inducing pneumothorax the depth to which it was necessary to penetrate the chest wall as far as the pleura; and in none of them was it deeper than 3 cm.; in the vast majority it was only between 1.5 and 2.5 cm.; in some less than 1.5 and in one even less than 1 cm.

**Technic of Insufflation.**—As soon as the lumen of the needle penetrates the costal pleura and there are no adhesions at the point of penetration, the tube leading to the manometer is opened and the fluid in the closed limb is seen to be sucked up. In some cases the suction is so pronounced that the fluid shoots up to the upper end of the tube and care must be taken that it is not aspirated into the pleura. Usually it is elevated between 1 and 6 cm. and oscillates. The patient is told to take a deep breath and it will be observed that during inspiration the negative pressure is more pronounced than during expiration. This oscillation is the only reliable indication that the lumen of the needle is in the pleural cavity, but at times there are observed slight oscillations when the needle reaches the costal pleura before puncturing it owing to the respiratory movements of the lung. But these oscillations rarely exceed 1 cm. and must not mislead us. Only when the negative pressure exceeds 3 cm. may we venture to let in the gas, and beginners should not do it with less than 5 or 6 cm. negative pressure.

**Manometric Hints.**—The manometer is to be watched, especially during the first operation. The following rules, based on the writings of Forlanini, Brauer, Saugman, Piéry, Balboni, Frederick C. Coley,<sup>1</sup> and personal experience, are useful guides.

*When the Lumen of the Needle is in the Thoracic Wall.*—As long as it is outside of the endothoracic fascia, the manometer rests at zero. When it reaches the endothoracic fascia, feeble oscillations, due to respiratory movements of the pleura, may be seen, but they are of slight amplitude, between 0 and 3 on each side of the manometer. They should not mislead us into the belief that the lumen is in the pleural cavity. The fact that there is no negative pressure proves this.

A slight negative pressure during inspiration, becoming less on expiration, may be produced when the point of the needle is really not in the pleural cavity at all, but pushing the parietal pleura before it. The indications are clear—the needle is to be pushed ahead guardedly till it punctures the parietal pleura.

<sup>1</sup> Lancet, 1915, ii, 469.

*After the Needle Passed the Parietal Pleura.*—When there are no adhesions there is at once seen negative pressure, 5 to 10 cm., and distinct respiratory oscillations, higher on the side of the manometer which is connected with the needle than on the side communicating with the outer air. If the patient holds his breath during inspiration or expiration, or the injection is stopped, the pressure remains negative or positive respectively.

But at times we meet with this anomalous condition: On passing the parietal pleura the fluid in the manometer rises high showing negative pressure of 10 cm. or more, but then it remains stationary. We know then that the lumen is in the pleural cavity, and that there are no adhesions, but we hesitate to proceed with the injection because there are no oscillations. It is clear that the lumen of the needle was for a moment between the pleural surfaces, but it has either pushed the visceral pleura ahead of it or entered the lung, or it has become clogged. In the former case slight withdrawal of the needle will reestablish oscillations; in the latter case we put the obturator into the lumen of the needle and clear it.

In case there are dense adhesions and the needle does not enter the pleural cavity, the manometer stays at zero and does not oscillate; or when slight oscillations are noted they are but one or two cm. and equal on both sides, or slightly positive.

When there are slight and yielding adhesions, there is feeble negative pressure, about 2 to 3 cm., and slight oscillations. Occasionally the adhesions yield and the negative pressure, as well as the oscillations, suddenly increases. But usually the pressure becomes positive soon after the introduction of some gas, indicating that a gas pocket has been created. During reinflations, sudden drops in the pressure, due to breaking up of adhesions, are more common than during primary inflations.

*When the Lumen of the Needle is in the Lung.*—The manometric indications will differ according to the structures the needle has penetrated. If it is in consolidated lung tissue there will be no change in the level of the fluid in the manometer; it rests at zero. If the lumen is in a bronchus or bronchiole, there is usually no negative pressure, but there may be slight oscillations of equal excursions. The amplitude of the oscillations will depend upon the character of the respiration, whether tranquil or labored. When the patient speaks, the respiratory effort with a closed glottis produces, while it continues, a greatly increased pressure, greater still on coughing. When the patient holds his breath, in inspiration or expiration, the manometric readings are again zero:

If while inserting the needle during the first attempt at inflation positive pressure is noted during expiration, it is proof that the lumen is in the lung, or in a bloodvessel. Occasionally it is found that the gas flows in freely, but the pressure in the manometer does not ascend. This is an indication that gas is escaping as it enters which could only

occur when the needle is in a bronchus and never when it is in the pleura. "If the key connecting with the nitrogen is quickly opened and immediately closed, allowing only a very minute quantity of nitrogen to flow in, the manometer then becomes positive, it is because the needle is in the lung" (Balboni).

*If the lumen of the needle is in a bloodvessel* there are no oscillations, but slight positive pressure may be observed; if some blood enters the needle, which is the rule, the pressure will be rising. When withdrawing the needle it will be found that it contains blood, and the patient may have hemoptysis.

**Injection of the Gas.**—With the assurance that the needle is in the pleural cavity the tube leading to the manometer is closed, the tube leading to the gas reservoir is opened, and nitrogen allowed to flow in by aspiration, or pressure when necessary. After 100 c.c. of gas have entered, the manometer is again consulted, and if still showing negative pressure, another 100 c.c. is allowed to flow in. It has been my habit never to exceed 300 c.c. during the first operation, although many do not hesitate to introduce two and even three times as much, and some even attempt to secure complete collapse of the lung during the first operation. Murphy advises the introduction of 200 cubic inches (3000 c.c.) at the first operation, while Forlanini now advises only 200 to 300 c.c. Clinical experience seems to favor smaller quantities as safer, and many unpleasant, often dangerous, symptoms are thus avoided. To change quickly the relations of the thoracic viscera is dangerous. Moreover, when adhesions are present, they may be forcibly torn apart and cause trouble. When extensive and dense adhesions are present, it is often impossible to introduce more than 100 to 200 c.c. of gas, and the chances of finally securing a complete collapse of the lung are rather slim.

On the completion of the operation the needle is quickly withdrawn and the index-finger of the left hand placed over the point of the puncture and some pressure applied with a view of preventing cutaneous emphysema. Finally the small wound is sealed with some cotton and collodion and the patient is warned against coughing, which he is to avoid as far as is within his control. I find a dose of morphin or codein is useful for this purpose. It has been my rule to send the patient to bed for twenty-four hours after the first operation, irrespective of his general condition.

**Method in Urgent Cases.**—In urgent cases, as in copious and uncontrollable pulmonary hemorrhages, and when no apparatus and tank of nitrogen are at hand, we may resort to Murphy's method which he describes as exceedingly simple: "Take an ordinary hypodermic needle, rub the sharp point dull on a brick, cover the butt end of the needle with cotton, which will serve as a filter of the air that is to enter, then insert the needle into the pleura at the point of election for the production of a pneumothorax. The skin should have been painted with iodine and punctured with a tenotome. The idea is to let the air

enter the pleural cavity through a needle, the cotton filtering it as it enters, thus producing a pneumothorax. The finger placed over the but end of the needle serves as a valve. As the patient inspires the finger is lifted off the needle to allow the air to enter, and on expiration the opening is closed with the finger. In that manner you can pump the pleural cavity full of air to any desired degree of compression. If the patient becomes too cyanotic, or if the breathing is embarrassed, lift the finger from the needle and allow a little air to escape. The procedure is now reversed. Close the end with the finger on inspiration and remove the finger on expiration, so that air will be pumped out instead of in."

**Technic of Refilling.**—The introduction of a few hundred cubic centimeters of nitrogen does not collapse or immobilize the lung. This must be accomplished gradually by further inflations. In cases with free pleuræ this is a simple matter considering that a pocket with gas has been already created and the needle can be easily introduced into it. For this reason it is best to do the second inflation in the neighborhood where the first puncture was successfully made, so that it enters the gas pocket, and only exceptionally is another place chosen. In the latter case we are guided by the same principles as during the primary puncture.

One thing is to be remembered: The manometer is always to be consulted before the gas reservoir is opened and, in case no respiratory oscillations are seen, the stillete is to be inserted into the needle on the assumption that the lumen may be clogged, which is often the case. If no oscillations are even then observed, the needle is to be withdrawn and reinserted in another place. Accidents have happened during later inflations just as during primary operations.

The quantity of nitrogen introduced during refills depends on the case. My way has been to introduce between 300 and 600 c.c. at the second and 800 to 1200 at the third operation, provided the patient bears it well. But when I find embarrassment of the circulation, dyspnea, or pain in the chest, I proceed slower and am satisfied with 300 c.c. given every other day till complete collapse is attained in two or three weeks. We are also to be guided by the final pressure after each inflation. In many cases we get positive pressure after several hundred cubic centimeters of nitrogen have been introduced, although there is no complete collapse of the lung. We often meet with cases in which the gas opens but a small pocket in the pleura and when this is filled the negative pressure decreases or vanishes. When oscillations are good the pressure may be increased guardedly, consulting the manometer after each 50 or 100 c.c. have entered. Saugman, whose experience is unexcelled, found that if the gas does not pass with 10 to 15 cm. water pressure the case may be given up, because higher pressure will meet with failure.

At times it is noted that during a refill the pressure suddenly sinks. This is an indication that some adhesions have yielded or, which is

fortunately exceedingly rare, that the lung has ruptured and the gas escapes from the pleura into a bronchus. This may occur when the nitrogen is introduced under high pressure and the patient coughs vigorously.

My experience coincides with that of Saugman to the effect that it is best that, during the first few fillings, the final pressure should not exceed 0.5 to 2 or 4 cm. of positive manometric pressure. The condition of the patient, as well as his reaction during the succeeding few days should, however, be our guide. We must always watch whether our aim is not attained with a low pressure, and in many cases 0.5 to 1 cm. above zero is sufficient. Forcible inflations involve rapid dislocation of the mediastinum and injury to the other lung. We must bear in mind that it is not always imperative to compress the lung. In most cases affording rest to that organ by immobilization is sufficient to give relief, and this can be attained without high intrapleural pressure. But in case the patient is not improving, his cough, temperature, expectoration, etc., are not influenced favorably, the pressure is carefully and guardedly increased. A final pressure of 10 to 15 cm. of water is too high, though many authors state that they have resorted to it in some cases. Of course, as a rule, the gas is quickly absorbed and within a few days the pressure drops so that the embarrassment of the respiration and circulation is ameliorated. The great problem is the cases in which only an incomplete pneumothorax has been created and the stiff, unyielding walls of cavities, or dense pleural adhesions, prevent the compression of the part of the lung which we aim to collapse. Saugman and Forlanini have not hesitated to increase the pressure in these cases to 30 and even 40 cm., and they were occasionally rewarded by finally attaining a complete pneumothorax.

**Frequency of Refilling.**—After complete collapse of the lung has taken place the frequency of the refillings is diminished. In some patients the gas is absorbed slower than in others and we are unable to say in advance who is likely to need frequent refills and who is likely to need infrequent refills. It seems that those walking around absorb the gas sooner than those who remain in bed. Primarily the guides for the necessity for refills are the general condition of the patient and secondarily the findings on physical examination. An elevation of temperature, if not due to an impending or actual pleural effusion, is often removed by a refill. The same is true of cough and expectoration. In those who had the lung completely collapsed, there is a complete absence of breath sounds and adventitious sounds; a return of these is an indication that refilling is necessary. The fluoroscope is, however, the best guide. But I want to repeat that dyspnea and tachycardia, which are often caused by excessive pressure in the pleural cavity, are to be guarded against.

**Symptoms.**—The acute and urgent symptoms of spontaneous pneumothorax are never seen in the artificially created pneumothorax, excepting, of course, when the lung is penetrated and the spontaneous

variety complicates matters. The pain, dyspnea, cyanosis and collapse are never encountered. In fact the majority of patients who have overcome the fear for the operation are ready and well able to leave their beds immediately after the operation and attend to their affairs. The slight difficulty in breathing, seen in some cases at that time, is usually objective, the patient protesting that he feels well although he evidently suffers from air hunger of some degree. But

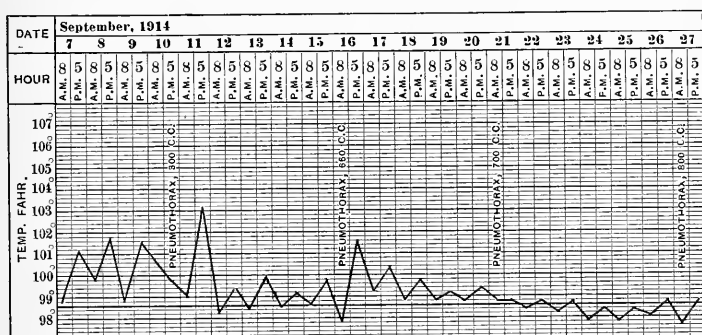


FIG. 87.—Showing the influence of therapeutic pneumothorax on the temperature.

even this disappears within a couple of days, as has already been mentioned. Only in rare instances, when the gas separates adhesions by high pressure, does the patient complain of pains in the chest which are, as a rule, trifling.

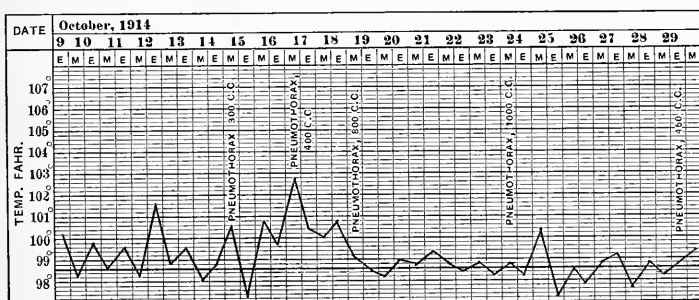


FIG. 88.—Showing the influence of therapeutic pneumothorax on the temperature.

In febrile patients the effects of the pneumothorax are usually striking, especially when complete collapse of the lung is attained. The fever disappears and, in successful cases, does not return unless there is some complication. The temperature charts (Fig. 87) distinctly show the effects of collapse on the temperature. In some cases it is noted that the fever increases  $1^{\circ}$  to  $3^{\circ}$  F. for twenty-four hours after each insufflation (Fig. 88) just as is the case with the reaction after

an injection of tuberculin. This is probably caused by increased toxic absorption, owing to the compression of the diseased lung. In case an increase in the temperature, lasting several days, is noted during the treatment, we may look for some unpleasant complications, especially a pleural effusion. When the pneumothorax does not reduce the temperature, we may consider the treatment a failure in this particular case. With the disappearance of the fever, the night sweats vanish and this gives the patient great relief.

The appetite improves in successful cases, and with this the lost strength is gradually regained, and the languor, which is such a strong clinical feature of the disease, is replaced by a feeling of well-being.

It is noteworthy that, in spite of the improvement in the general condition of the patients, the gain in weight is not a constant phenomenon in artificial pneumothorax. As long as the general condition of the patient is good, and the loss in weight inconsiderable, it should not trouble us. When, however, the loss of weight is considerable and general symptoms, such as fever, sweats, anorexia, etc., make their appearance, we may first try to reduce the pressure in the thorax, and if this does not ameliorate the condition, the treatment may have to be given up.

Great relief is usually obtained in patients who suffer from severe coughing spells which keep them awake during the night. This is especially true of unilateral cases in which a large cavity is emptied by compression. After the first three or four inflations it is constantly observed that the amount of sputum expectorated is augmented because the pressure exerted by the gas empties cavities and bronchi of their contents. After the lung has completely collapsed, or the cavities have been emptied in partial pneumothorax, the quantity of sputum diminishes, and in unilateral cases, expectoration ceases altogether. In many cases tubercle bacilli are not found in the sputum after the lung has been compressed for two months.

More striking than the improvement in the general condition is the cessation of hemoptysis when the first inflation is made in a case of hemorrhagic phthisis in which the patient is in constant dread lest the hemoptysis recur. We can assure him that he is safe in this regard. In hemoptysis pneumothorax acts as a hemostatic like the tampon in uterine hemorrhage. If during the treatment blood-spitting occurs, despite the collapse of the lung, we may be satisfied that the blood comes from the untreated lung.

In many, though not in all cases, there occurs some dyspnea during the operation or immediately after. But this is, as a rule, transitory. In fact, when the dyspnea is due to fever or toxemia it disappears after the induction of pneumothorax. If excessive pressure is permitted to prevail in the treated pleura, dyspnea is likely to occur which is usually transitory. The absence of the dyspnea, despite the cutting of the breathing area in nearly one-half, is not surprising because, in pneumothorax and in pleural effusion, a reduction of 66 per cent.



of the respiratory area does not materially alter pulmonary ventilation, nor the chemistry of respiration, provided the patient is at rest.

**Physical Signs.**—Recalling the physical diagnosis of spontaneous pneumothorax as given in text-books, we are surprised that most cases of artificial pneumothorax do not show any of the supposedly pathognomonic signs. Thus, tympany is not a constant sign, and in some cases the treated side of the thorax is simply hyperresonant and, in contrast with the untreated side, only shows a tympanitic overtone, because of the vicarious emphysema in the latter, which is hyperresonant or even tympanitic on percussion. It is hazardous to diagnose pneumothorax on signs obtained by percussion alone. The only feature that may give a clue is displacement of the heart, especially in cases of left-sided pneumothorax, in which even a small amount of gas may shift the heart to the right.

On auscultation we find in cases with complete collapse of the lung total absence of breath sounds, as well as of any rales which may have been audible before the gas was introduced. In these cases we may be guided by the auscultatory findings as to the necessity for refilling. When the breath sounds return it means that quite some portion of the gas has been absorbed and must be replaced at once. In cases in which the lung has been collapsed, but large bronchi have remained active, we may hear distinct and exquisite amphoric breathing or distinct metallic breath sounds, which shows that the teaching of some text-books to the effect that the amphoric phenomena in spontaneous pneumothorax are invariably due to bronchopleural fistulæ is erroneous. They are evidently due to sounds originating in the bronchi which reverberate in the air filled pleural cavity.

The progress of the pneumothorax can usually be followed by noting the increase in the area of the thoracic surface over which there is either absence of respiratory sounds or amphoric breathing after each filling, till finally the complete lung is collapsed and all breath and adventitious sounds disappear.

**Complications.**—Not all cases of induced pneumothorax run a smooth course during the period of treatment. Complications may arise during the operation or immediately after, and while the patient goes around with a collapsed lung. Of the former, collapse, pleural shock, or pleural eclampsia, pains in the chest, and subcutaneous emphysema are worthy of consideration; of the latter pleural effusion is the most important.

**Pleural Shock.**—Pleural shock may be of various degrees. The mild forms manifest merely an increase in the rate of the pulse and respiration, pallor, dyspnea, etc., which pass within a few minutes or an hour. I have met with it several times; in one patient it occurred consecutively during the first four inflations and I am inclined to attribute it in a great measure to his fear for the operation. In one of my cases the shock was quite severe, even alarming, yet it passed away within half an hour. Several authors have reported fatal cases.

The etiology, especially of the fatal cases, is not clear. Forlanini, Saugman, and others are inclined to attribute it to reflex spasm of the cerebral or cardiac bloodvessels. It has been observed that thoracocentesis for any purpose may cause collapse or even death on very rare occasions. Brauer is inclined to attribute the symptoms of shock to gas embolism in most cases and says that the fact that it is usually transitory does not exclude gas embolism. But pleural shock may occur without any gas inflations. James A. Lyon<sup>1</sup> mentions a case occurring while injecting novocain into the pleura.

That this accident is comparatively rare is evident from Forlanini's figures to the effect that operating on 134 patients, not including those in whom he failed to produce a pneumothorax, and making more than 10,000 operations, he met with pleural shock only twelve times. Among the 150 inflations made at the Montefiore Home we observed it but once to be sufficiently severe to cause some alarm.

**Gas Embolism.**—When the manometer is not properly consulted, it is said that at times even when the most careful technic is followed, gas may enter a bloodvessel and be carried away to any part of the body and produce an embolism. Usually one of the pulmonary veins is entered, and it is well known that negative pressure prevails in these vessels. Brauer maintains that one of the veins around an infiltrated area of lung tissue or of pleural adhesions may be penetrated by the needle and gas introduced into the circulation. The nitrogen is carried into the left heart, then into the aorta, whence it may travel into the coronary arteries or the cerebral vessels. Experimental researches have not been uniformly confirmatory of this theory, and clinically the symptoms of embolism have been observed in some cases even when no nitrogen was allowed to enter through the needle—merely after introducing the needle.

Wolff-Eisner,<sup>2</sup> while agreeing that in most cases it is due to gas embolism, says that there are some in which thrombi are responsible for the symptoms observed. They are derived from the vessels around or within the pulmonary or pleural lesion and dislodged by the needle. However, it must be emphasized that symptoms of gas embolism are not exclusively encountered in the primary operations, but have been met with during refills.

The symptoms are collapse, rapid pulse, irregularity of respiration, numbness, giddiness, inequality of the pupils, hemiplegia, etc. In some rare cases death has occurred without warning. I have been fortunate in not having met with a single case of this kind in my practice. Of course prophylaxis is to be the chief aim while operating, and one who does not permit the gas to flow into the chest without considerable oscillations of the manometric column is hardly likely to meet with a case. Fatal cases have, however, been met by the best and most experienced operators.

<sup>1</sup> Boston Med. and Surg. Jour., 1914, clxxi, 329.

<sup>2</sup> Die Prognosenstellung bei der Lungentuberkulose, Berlin, 1914, p. 498.

**Pains.**—Pains in the chest are felt by the patient occasionally during the operation. At times while introducing the needle as far as the costal pleura and before penetrating it exquisite pains are felt which promptly disappear as soon as the pleura is punctured. This can be prevented by proper anesthesia of the pleura with novocain or cocain. Very often after the introduction of the gas, pains are felt in the chest for twenty-four hours, due to breaking up of adhesions, especially when high pressure is applied. They are not at all unbearable and need no treatment. Abdominal pains may result from lowering of the diaphragm by the intrapleural gas pressure, but this is also transitory and needs no treatment.

**Spontaneous Pneumothorax.**—Spontaneous pneumothorax may occur when the needle lacerates the visceral pleura, or when a superficial lesion or cavity of the lung breaks through after the pleural sheets are separated by the gas. Forlanini has met with 9 cases of this kind. Floyd<sup>1</sup> and Webb<sup>2</sup> mention it. Alfred Meyer<sup>3</sup> mentions a case in which it occurred while preparations were being made for the induction of an artificial pneumothorax. Of course, when this complication is due to the entry of the needle into a cavity, or even a caseating part of the lung, pyothorax with its concomitants are likely to be the result. In fact, such cases have been reported.

According to W. Parry Morgan, "spontaneous" pneumothorax is more often produced while inducing an artificial pneumothorax than is generally appreciated. This is confirmed by the occasional cases met with in which the treatment is abandoned after a futile attempt to introduce gas into the pleura, and a collapsed lung is then discovered. Again, a radiogram of the chest taken after the first operation usually shows evidence of more gas in the pleural cavity than has been introduced from the reservoir. While it is common experience of those using the method that gas can be detected after 200 to 300 c.c. have been introduced, it has been Morgan's experience that if the visceral pleura is not injured the gas cannot be detected until considerably more than 300 or 400 c.c. have been introduced. He concludes that when a pneumothorax is visible in the fluoroscope after introducing 300 or 400 c.c. of nitrogen, we have justification for the conclusion that radiographic demonstration of a pneumothorax after the introduction of such a quantity of gas is achieved only by this being largely supplemented by leakage from the lung.

**Emphysema.**—The infiltration of gas into the subcutaneous tissue of the thoracic wall around the point of puncture is very frequently observed, especially in those operated upon by the Brauer method. In the vast majority of cases it is due to the high pressure of the gas in the pleural cavity, supplemented by cough, and the nitrogen works its way along the track of puncture. It is readily recognized by the

<sup>1</sup> Boston Med. and Surg. Jour., 1913, clxix, 713.

<sup>2</sup> Trans. National Assn. Study and Prev. of Tuberc., 1914, x, 101.

<sup>3</sup> *Ibid.*, p. 112.

crepitation elicited on palpation, and is of little significance—passing away spontaneously within three or four days or at most a week, and further inflations are not contra-indicated while it is present. It may be prevented by using thin needles and warning the patient against coughing, or administering some sedative like codein immediately after the operation. It has occurred in about one-half of my cases after the first or second operation and rarely after later inflations.

Of more serious import is emphysema of the deeper tissues of the thorax which, fortunately, occurs only rarely and may be avoided by careful technic. It is usually due to the introduction of nitrogen into the subpleural tissue before the lumen of the needle has penetrated the costal pleura. As was shown by Brauer, Spengler, and others, deep emphysema may also be due to leakage from the pleural cavity through the wound made by the needle, the gas being pressed by the intrapleural pressure or the respiratory movements especially during cough, into the extrapleural tissues. Saugman is of the opinion that this may even occur without excessive intrapleural pressure although the latter enhances the chances of its occurrence. The gas works its way along the path of the vessels to the posterior mediastinum and thence along the vessels and trachea up to the neck, where we may discover it by the crepitations along its anterior aspect. It is noteworthy that it is often felt earlier on the untreated side of the neck, which Saugman believes is due to posture. Rarely the emphysema may extend along the vessels to the face, shoulder, arm, and forearm. It may be severe enough to cause dysphagia and pain wherever it occurs. But the ultimate outcome is always favorable—it disappears within a few days or a week. It has occurred in two of my cases and, barring the little inconvenience it caused them, it was of no significance. Saugman, who had considerable experience with deep emphysema, states that in the patients in whom it occurs there are but few chances of inducing a complete pneumothorax because of the gas leakage.

With the abdominal emphysema which has been described by several authors, I have had no experience. It may occur when the needle is inserted along the lower margin of the chest and the diaphragm happens to be unduly high, which is not unusual in pulmonary tuberculosis. The lumen of the needle may then reach the peritoneum, between the diaphragm and the stomach or liver. It is to be remembered that there also the manometer will show negative pressure, oscillating with the respiratory movements. Saugman points out that it is difficult to distinguish these oscillations from those seen when the needle is in the pleural cavity, but if it is carefully watched it will be observed that when the needle is in the pleural cavity the negative pressure is stronger during inspiration, and the reverse is true when the lumen of the needle is in the peritoneal cavity.

**Pleural Effusions.**—The most frequent and serious complication of artificial pneumothorax is pleural effusion in the course of the treat-

ment. Its frequency varies with the different reports by various authors. Some report as high as 60 per cent. of cases, while others have met with it less frequently. Some are inclined to attribute it to "colds" or to "rheumatism," etiological factors which are open to question. Others have stated that it is usually due to infection during the operation and maintain that when asepsis is rigidly observed, effusions are rare, which does not hold, because effusions have been met by the most careful of operators. Floyd says that where injections are very frequent and small amounts of nitrogen are given at a time it is more likely to occur than where the interval is of some duration. Bullock and Twitchell<sup>1</sup> say that it may be prevented by using warm nitrogen. Faginoli<sup>2</sup> considers the nitrogen as a foreign body which irritates the serous surface of the pleura, predisposing it to disease. It becomes a *locus minoris resistentiæ*, and inflammation occurs more easily than in ordinary cases of phthisis. Klemperer's<sup>3</sup> explanation is more plausible: Disease processes which reach the surface of the lung and the visceral pleura, cause adhesions in patients with normally superimposed pleural sheets, but in pneumothorax with separated pleural sheets exudative inflammations are the result. Ruptures of adhesions which lay bare tubercular foci in the pleura may also be instrumental in infecting the complete serous surface. Bullock and Twitchell<sup>4</sup> consider these exudates a response to irritation by the foreign body, the gas. "The secretion of a fluid by the pleura is as natural a phenomenon as that of tears by the conjunctiva. If the tear duct is occluded, the tears will overflow upon the cheeks. When the mechanism of the pleura is in perfect working order as to secretion and absorption an excess of fluid is never found; but we certainly know that as pneumothorax is protracted the absorption properties of the pleura became more and more impaired." The fact that the fluid usually contains lymphocytes and is pathogenic to animals is conclusive proof of the tuberculous origin of these effusions.

The diagnosis is difficult at the onset. In most cases there is a rise in the temperature, though at times it may pass afebrile; but there is no chill. The fever is hectic and may reach 103° F. and higher. There is also a rise in the intrapleural pressure which cannot be accounted for by the insufflations, and the manometric oscillations are diminished. Groco's triangle can be made out when the effusion is considerable, though Faginoli says that it is always absent. Small effusions are often very difficult to diagnosticate, and even the fluoroscope may fail to reveal them. They are especially difficult to discern in radiograms which have been taken with the patient reclining, for obvious reasons. When more or less copious, the usual signs of pleural effusion

<sup>1</sup> Amer. Jour. Med. Sci., 1915, cxlix, 848.

<sup>2</sup> Riv. crit. di clin. med., 1912, xiii, 678, 694.

<sup>3</sup> Berl. klin. Wehnschr., 1911, cxlvii, 372.

<sup>4</sup> Amer. Jour. Med. Sci., 1915, cxlix, 848.

are present plus the succussion sound and the splash, which are at times annoying to the patient.

The effects of the effusion depend on whether they are of a toxic nature or not. In the former case there is prolonged fever of a hectic type. Simple effusions, as has been pointed out by von Muralt, are rather salutary phenomena and may have a good effect on the general and local condition of the patient by the antibodies they produce. Faginoli does not agree with this view and says that in the end effusions interfere more or less with the favorable outcome of the case. Saugman also states that in the majority of cases it is a rather disagreeable complication, which is in agreement with my experience. The patients who have had effusions have not done as well as those without this complication.

As long as there is no fever and no cardiac embarrassment, the effusion should not be interfered with, because it keeps the lung collapsed, and this is just at what we aim with the treatment. But in cases in which the fever is high it may be necessary to withdraw part of the fluid and replace it with nitrogen. In some cases I have applied autoserotherapy—withdrawing 10 c.c. of fluid and reinjecting it subcutaneously, and am under the impression that it enhances absorption. We must always watch these exudates. In case they are absorbed too rapidly, the lung reëxpands and may form adhesions, thus preventing its further collapse by the gas inflations. Pyopneumothorax is not rare. It is invariably fatal sooner or later.

**Active Lesions in the Untreated Side.**—Extension of the disease in the other lung is perhaps the most disheartening complication during the treatment. It has been stated that it may be caused by an attempt to collapse the more affected lung too quickly; the purulent matter is squeezed out rapidly, and it travels along the bronchi to the other side of the chest, producing pus embolisms. It has also been attributed to excessive pressure in the pneumothorax. It has occurred in some of my cases and in none could I attribute it to these causes. In some of my cases there was a hemorrhage from the untreated lung, but it soon ceased. There have been reported cases in which one side of the chest was treated by a pneumothorax and the lesion was cured, but subsequently a new lesion flared up in the opposite lung, which was also treated by a pneumothorax. This indicates that the collapse and compression of a lung do not necessarily impair its function permanently.

**Indications.**—Forlanini at first urged that only far advanced cases of phthisis for which everything had already been tried, but no relief was obtained, should be given artificial pneumothorax. As a *conditio sine qua non* it was insisted upon that the lesion must be strictly unilateral, and that any involvement of the other side of the chest is a contra-indication to the treatment.

**Factors Entering into the Selection of Cases.**—*The Form and Stage of the Disease.*—There are numerous cases of phthisis which are doing

well and even recover with or without any treatment, medicinal, specific, climatic, or institutional, and it is, of course, not advisable to subject them to the operation with its potential complications. This is true of mild incipient cases and abortive tuberculosis. Fibroid phthisis runs an exceedingly chronic course; the pleura is often extensively involved, precluding the introduction of gas into the hemithorax most affected and cannot be treated by this method. This is also true of the most common forms of fibroid phthisis characterized by diffuse fibrosis all over both lungs, and it would be sheer folly to treat but one side of the chest. On the other hand, in the later stages of diffuse fibrosis, when excavations form in one lung, the question of pneumothorax is to be considered, provided, of course, that the pleura is free from dense and extensive adhesions.

It is the acute and progressive form of phthisis in which artificial pneumothorax finds its best indications and shows the best and most striking therapeutic results. In the group of cases known as galloping consumption, in which the patient is carried off within three to six months by a rapidly progressing infiltration, caseation and excavation, there are many who can be saved by the induction of pneumothorax. It is fortunate that dense pleural adhesions are exceptional in these cases and a pneumothorax can easily be induced. The results are often astonishing—with the collapse of the lung, the tachycardia, fever, night sweats, cough, expectoration, etc., disappear, and within a few weeks the patient is reinvigorated and may continue to gain in weight and strength indefinitely.

Another group of cases in which artificial pneumothorax renders excellent service are those which have recurrent, copious, and uncontrollable hemorrhages. While, when afebrile, the patients are not in grave danger, and death due to exsanguination is rare, yet our efforts to prevent recurrence of hemorrhage after one has been stopped by keeping the patient in bed for several weeks are often futile, and he, as well as those around him, are discouraged. I have had some patients who had to remain in bed for two or three months with slight but protracted hemorrhages, one following another. With the induction of a pneumothorax, provided we succeed in completely collapsing the lung, we have an excellent means of controlling the hemorrhage, to prevent its recurrence, and in addition to giving the tuberculous focus an opportunity to heal.

It is obvious that only one lung may be compressed while the second must be left to carry on the functions of respiration, and that it is useless to combat a lesion in one lung while the disease is smouldering or progressing in the other. For these reasons it has been found advisable to apply pneumothorax only in unilateral cases. But as a matter of fact, in more or less advanced phthisis unilateral lesions are hardly, if ever, met with. Klemperer says that he hardly knows of a case in which only one lung was extensively involved and the other remained free from the disease in the anatomical or bacteriological

sense. Clinical experience is supported in this regard by autopsy findings. Inasmuch as strictly incipient cases are not to be treated by this method for reasons already stated, it is evident that in nearly all cases in which pneumothorax is indicated there will be found signs of involvement of both lungs and we must be satisfied with mild or moderate lesions in the untreated side.

In practice we find that in the vast majority of moderately and far-advanced cases the lesions are extensive and active in one lung, while in the opposite there is limited involvement, or signs of quiescent or healed lesions. Though not strictly unilateral, these cases can be successfully treated by pneumothorax, if not prevented by pleural adhesions.

It is interesting that careful clinical and pathological observations have shown that only exceptionally is the untreated lung unfavorably affected. In spite of the increased functional activity because of the vicarious work it is compelled to do, the lung usually remains in the same condition as it was before the opposite lung was collapsed. The vicarious emphysema which is, as a rule, produced, increases the size and dilates the alveoli and bronchioles, thus permitting as much air to be passed through as before, when both lungs were active. Forlanini, Saugman, Robinson, Floyd, Hamman, von Adelung, Lyon, and many others have reported cases in which active lesions in the untreated lung have improved or healed after a pneumothorax was induced in the more affected side. The factors operative in such cases are not well understood. Carpi<sup>1</sup> has pointed out that amphoric sounds and rales are often altogether transmitted from the more affected side and that diagnosis is very difficult. On the other hand, the increased blood supply may have something to do with it. The diminution in toxic absorption from the ulcerating and excavated lung may give the patient a chance to recoup his natural reparative forces, unhampered by the toxemia from extensive suppurating areas. However, this is not the rule. In some, lesions in the untreated lung flare up and extend, as has happened in some of my cases. In some of my cases copious hemoptysis even occurred from the untreated lung.

Recently Forlanini and many others have argued that all advanced cases should be given an opportunity to benefit by artificial pneumothorax. In far-advanced, bilateral, or "hopeless" cases one side as is a rule extensively involved, while the other side shows only limited involvement, though the lesion may be evidently active. In such cases it is urged that the more affected side should be treated on the principle that there is nothing to lose and everything to gain. Forlanini's experience has taught him that when the untreated side has but a limited, even though active focus, the chances of a success are better than would be expected *a priori*. When both sides are extensively affected the chances of recovery are slim indeed, but improvement in

<sup>1</sup> Gazz. med. ital., 1911, lxii, 461, 473.



the general condition may be anticipated and a prolongation of life is not unlikely. At times, Forlanini says, we may be astonished that even such patients are cured. In most cases the removal or diminution of toxic absorption gives the patient an opportunity to muster his natural forces of resistance and comfort, often superior to that obtained in operative procedures for incurable cancer of the stomach may be procured.

There is another important point to be borne in mind: We are not always able to ascertain positively whether the lesion in the less affected side is active, quiescent, or even healed. Rales and amphoric breath-sounds heard over a given area of the chest wall are not always autochthonous, but may be in fact transmitted by conduction from the opposite side, and this is at times very difficult to differentiate, as was already mentioned. Indeed, perfect symmetry of location of rales, especially on both sides of the spine in the upper part of the chest posteriorly should always excite suspicion that they may be transmitted, and on the side on which they are weaker it is probably so. During and after pulmonary hemorrhages also there are often heard rales all over the chest which disappear in the unaffected side within several days, but when audible they give the impression that both lungs are extensively involved. Skiagraphy is of little, if any, assistance in clearing up many of these cases.

Some French and Italian authors have suggested "diagnostic pneumothorax" in cases in which we are uncertain whether the disease is active in both sides. The more affected pleura is inflated with gas and the opposite lung is watched. In cases in which the physical signs of disease are of the transmitted kind, they disappear soon after the lung is collapsed. But in case they persist in spite of a complete pneumothorax and the general condition of the patient is aggravated, the pneumothorax is allowed to be absorbed or, in more urgent cases, the gas is aspirated and the lung permitted to reexpand.

There are some who believe that even incipient cases ought to be treated with pneumothorax. Among these may be mentioned Murphy,<sup>1</sup> Lemke, Bullock and Twitchell, Gray,<sup>2</sup> Forlanini, von Adelung, Piéry, and some others. Murphy and Kreuscher say: "Is it well to wait until the outlook is so desolate? Is the lung collapse such a desperate operation as to be used only as a last resort?" With this I am not in agreement. If the treatment lasted only a certain and limited time, the patient could be informed of the details and given the choice. But inasmuch as we are not in a position to give the patient definite information as to the probable duration of the treatment, and a large proportion of these cases recover under the old and tried methods of treatment, we should not subject mild incipient cases to the dangers, complications, and duration of pneumothorax. I still hold that only progressive or hopeless cases are to be given this treatment.

<sup>1</sup> Interstate Med. Jour., 1914, xxi, 266.

<sup>2</sup> Illinois Med. Jour., 1913, xxiv, 201.

**Contra-indications.**—To some extent the contra-indications have already been given while speaking of the indications, but there remain yet to be discussed certain conditions which preclude the induction of an artificial pneumothorax, mainly those depending on the clinical form of the disease, the coexistence of extrathoracic tuberculosis and of other diseases. Because pneumothorax only acts locally on the treated lung, acute miliary tuberculosis, in which both lungs are usually equally involved, is not suitable for this treatment. Fibroid phthisis with extensive pulmonary emphysema is not suitable for this mode of treatment, excepting when in addition to the emphysema there is a localized suppurating excavation which is the cause of fever, sweats, cough, expectoration, etc., undermining the patient. An artificial pneumothorax may be applied as a palliative measure.

The most important forms of extrathoracic tuberculosis which complicate phthisis are laryngeal and intestinal involvement. Clinical experience has shown that pneumothorax may relieve these complications to an amazing extent. It appears that when the tuberculous toxemia, due to an extensive focus in the lung, is removed by a pneumothorax, the laryngeal and intestinal lesions often improve and there are even some cases in which complete cure was obtained of both the lung condition and the extrathoracic lesions. A. de Gradi,<sup>1</sup> Zink,<sup>2</sup> von Adelung, and others have reported such cases, and Forlanini speaks of them, though he confesses his inability to explain them. Conceding that the chances of cure are remote, laryngeal and intestinal complications should not deter us from applying pneumothorax if the case is otherwise suitable, on the principle that there is nothing to lose and everything to gain.

Diseases of the heart, bloodvessels, and kidneys have been found to materially lessen the chances of recovery with an artificial pneumothorax and are therefore mentioned as contra-indications to the treatment. They are all accompanied by disturbances of the circulation and the patients do not bear the deprivation of the breathing area of a complete lung. Forlanini, however, has found that when compensation is good, pneumothorax may be induced with same chances of success. Some object to the production of a pneumothorax in persons over forty years of age.

Diabetes has not been found to interfere with the successful outcome of an artificial pneumothorax, and the same is true of pregnancy. There have been reported several cases in which pneumothorax was induced in pregnant women who went on to term, were delivered of healthy infants, and continued under the treatment. In one of my cases the woman was six months pregnant when a pneumothorax was induced. The effect on the lung was excellent, complete collapse was attained and the general symptoms completely disappeared. The temperature chart (Fig. 89) shows clearly the effect on the fever

<sup>1</sup> Gazz. med. ital., 1910, lxi, 281.

<sup>2</sup> Beitr. z. Klinik d. Tuberkulose, 1913, xxvii, 155.

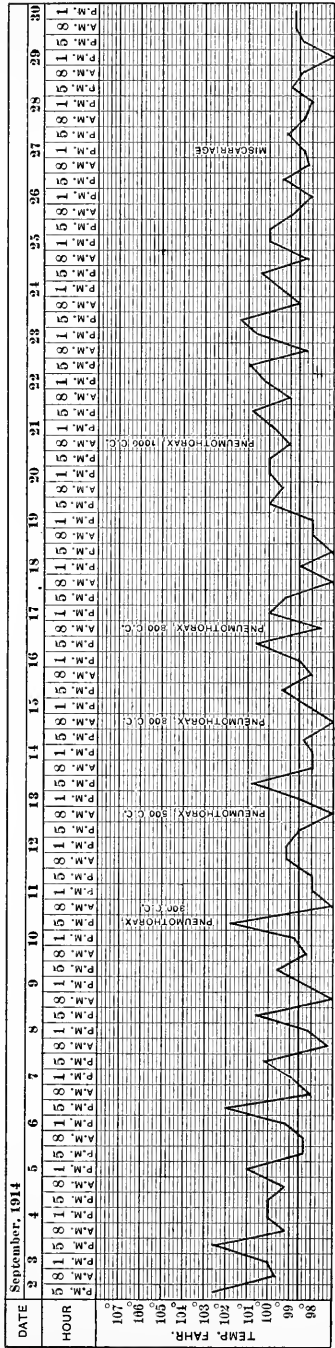


Fig. 89.—Effects of artificial pneumothorax on the temperature in a phthisical pregnant woman. The patient recovered.

which has been so far permanent for two years. But she miscarried four weeks after the first inflation of gas. It is noteworthy that the temperature and the general condition of the patient were not influenced by the miscarriage.

**Pleural Adhesions.**—These are, strictly speaking, not necessarily contra-indications to the induction of a pneumothorax, but they are hindrances to its successful accomplishment. In many cases no nitrogen at all can be introduced because of extensive and dense adhesions and, after several punctures are made, the case is given up as unsuitable for treatment. Frequently an area is found which is free and some gas is introduced, but further attempts to introduce a sufficient quantity to completely collapse the lung meet with failure. This failure may be of various degrees. In some the pleura is free only over a small area and a small pocket of gas can be made, while the rest of the pleura is adherent. No improvement in the condition can be expected and the treatment must be abandoned. Pleural adhesions often interfere with the treatment in a peculiar way. The pleura is free all over the chest except its upper third, over the tuberculous lesion, where it is densely adherent. There may be a cavity in that location surrounded by stiff walls. The result is that while we succeed in collapsing the lower two-thirds of the lung, the part which is diseased and which we aim mainly at collapsing in order to expel the pus and detritus from the purulent cavity and thus prevent toxic absorption and bring about coaptation of its wall with a view of giving them an opportunity to cicatrize, cannot be collapsed and the disease keeps on its usual course. This is notably the case with old cavities having stiff fibrous walls which refuse to yield to the gas pressure. Many failures are due to this condition. Fig. 3, Plate XVIII shows a radiogram of such a case. In spite of all efforts to collapse the lung completely, the adhesions around the lesion prevented the collapse of the diseased part of the lung.

At times the pleural adhesions are not very dense, in fact slight adhesions are said to be present in practically all advanced cases of phthisis, and an increase in the pressure while introducing the gas breaks them up and success is finally attained—the lung is completely collapsed.

The proportion of cases suitable for the treatment is very small indeed. Among 210 patients admitted to the Montefiore Home we found only 22 which we considered suitable for the treatment. This is a rather high percentage and is partly due to the fact that strong efforts were made by me to find suitable patients outside of the institution and induce them to enter. Statistics of most writers seem to indicate that less than 5 per cent. of all cases that come under their observation are suitable for this treatment. Lemke<sup>1</sup> appears to be the only author whose clinical experience has been to the effect that he has had to abandon the operation in all but a small proportion of the

<sup>1</sup> Jour. Amer. Med. Assn., 1899, xxx, 959, 1023, 1077.

selected cases, because of pleural adhesions. Perhaps the reason is that he operated on incipient cases. Bernard<sup>1</sup> found among 628 patients only 22 in whom he thought pneumothorax was indicated, and among these he succeeded only in 6 cases in completely collapsing the lung, in 11 adhesions prevented the creation of a complete pneumothorax, and 3 refused to submit to the treatment. J. Courmont found among 352 patients only 31 were suitable. Among 110 apparently suitable cases only in 32 per cent. could Zink produce complete pulmonary collapse, and in 24 per cent. he failed to enter the pleura altogether because of pleural adhesions.

Saugman found that in 30 per cent. of the selected cases adhesions prevented the entry of gas into the pleural cavity. Even with Brauer's method, the proportion of failures exceeds 25 per cent. It must, however, be mentioned here that while in most cases complete collapse is best, a partial pneumothorax at times serves a good purpose, and many writers report excellent results when only creating one or more gas pockets in the pleura, and in two of my cases the improvement was remarkable under such conditions. Von Adelung even practises partial inflation of the two pleuræ *simultaneously* in bilateral cases, and he says that the results have thus far been apparently beneficial. To my mind this improvement can only be seen in chronic cases of phthisis in which the cavities have been surrounded by stiff walls of connective tissue, and which do not secrete any more. Exquisite amphoric breath sounds are heard over such cavities, but no rales. The excavations are not the cause of the general symptoms which disable the patient, but the more acute patches of infiltration in other parts of the lung are responsible for the fever, nightsweats, etc. Compressing these parts we may achieve good results. In these cases we hardly ever achieve a cure with pneumothorax, because the cavity cannot cicatrize or contract owing to the stiffness of its walls which, together with the pleural adhesions, prevents its collapse by the gas pressure. But they may be greatly relieved by a pneumothorax.

**Duration of the Treatment.**—The question how long the pneumothorax must be maintained in order to achieve a cure cannot be answered categorically; no rules can be laid down which will apply to all cases. In fact, considering that this method of treatment has been applied such a short time, there are few who have many cases under observation for from six to ten years, and even they have not agreed as to the usual duration of treatment of a successful case. The following principles are based on the experiences of Forlanini, Brauer, and Saugman, who have had patients under their care for many years, as well as on my own observations, which have not yet extended for a sufficient period of time to give definite conclusions.

It appears that we cannot count on less than two years in the most favorable cases, although I have had success within one year in several cases—the pneumothorax was allowed to be absorbed and there

<sup>1</sup> Le pneumothorax artificiel dans le traitement de la tuberculose pulmonaire, Paris, 1913.

occurred no relapse of the disease. But these cases are few. To my mind, the most difficult problem is to determine when the healing process has been completed, so that if the lung is permitted to reëxpand no active lesion will remain to flare up again by the respiratory movements. This, however, is difficult and, I believe, impossible to determine with any precision as long as there is complete collapse of the lung, and the general condition of the patient is good because of the collapse. Moreover, if we allow the pneumothorax to be absorbed too early there may not only be a relapse of the disease, but experience has shown that the pleural sheets are likely to adhere, and the fibrous bands prevent the formation of a new pneumothorax, if we find that this is indicated.

It is for this reason that whenever we decide to discontinue the treatment we must watch the patient carefully while the gas is slowly being absorbed, and if some symptoms appear, such as fever, cough, expectoration anorexia, tachycardia, etc., we must at once reinflate the pleura. Forlanini says that many patients require a pneumothorax "forever," which is undoubtedly true, and most authors who have had experience with this method of treatment for many years, and had opportunities to observe their cases for long periods of time, agree with him.

Saugman, who has treated numerous cases with artificial pneumothorax and observed them for many years, says that when only a partial pneumothorax has been created which, however, has had a good effect on the symptomatology of the disease the treatment must be continued for at least two years, often for a longer period, according to clinical indications; in some cases "forever." In cases in which complete collapse of the lung was attained, we may expect a successful termination in one year, and in some acute cases the treatment may be discontinued within one year. Forlanini, Brauer, and others have had in some cases good and even permanent results after six months' treatment. It is, however, better to continue for at least two years in all cases. In chronic cases we must consider two years as the absolutely shortest period of treatment, and in doubtful cases it must be prolonged for three and even four years. The inconvenience to the patient in having infrequent refills, four to six annually, is trifling considering that he can pursue his vocation, compared with the hazards of a relapse in case the lung is allowed to reëxpand too early. It is therefore better to continue the treatment for a year longer than to stop one month too early. If the disease is extensive it is advisable that the inflations should be continued over long periods of years, perhaps "forever."

**Results of Pneumothorax Treatment.**—We have seen that hardly 5 per cent. of cases of phthisis are suitable for pneumothorax treatment. In other words, even if all the cases subjected to the operation were cured, which is not the case, 95 per cent. of the sufferers from this disease are not suitable for the treatment.

In suitable cases, especially those running an acute course, the effect is often striking—the fever declines and with it the symptoms

of toxemia, etc. But in many cases the improvement is not permanent. One of the complications, like pleural effusion in more than 50 per cent. of these cases, again brings about fever and symptoms of toxemia, etc. In many cases we are finally compelled to abandon the treatment because after the pleural effusion, adhesions prevent the introduction of more gas. In others, a lesion in the untreated side flares up and gives trouble, as might be expected. In still others the lung is compressed all over excepting the upper third, where the main lesion is located, because there it is held by some dense pleural adhesions which cannot be separated by increased gas pressure. Autopsy experience teaches that often such plural adhesions can hardly be cut with a knife.

Under the circumstances the number of cases cured by this method is rather small. Statistics which can be considered reliable are not available, because hardly two authors have reported comparable material. Lemke and others treated incipient cases, which should not be done. Others treat only advanced strictly unilateral cases; still others confine the treatment to cases in which there is nothing to lose, etc.

This should not deter us from applying the treatment in all cases in which it is indicated. We must always bear in mind that in "hopeless" cases an artificial pneumothorax often saves life, gives comfort and in some even efficiency, which cannot be obtained by any other method of treatment practised at present. All our cancer surgery, of which some surgeons speak with justifiable pride, does not give results comparable with artificial pneumothorax in hopeless cases of phthisis. No surgeon hesitates in performing the operation of gastrostomy for cancer of the esophagus or stomach, knowing that in all probability the patient will not survive three months. Palliative enterostomies, tracheotomies, etc., are performed with confidence that the best is done; even when life is not saved, comparative comfort is given during the last days of life of the unfortunate patient. In hopeless cases of phthisis artificial pneumothorax does much more than this palliative surgery: it removes the symptoms which make the life of the patient miserable—the cough, the expectoration, the fever, the nightsweats, anorexia, hemoptysis, etc.; reinvigorates him, and in many cases renders him efficient at his calling or even to do some light manual labor, irrespective whether he is ultimately cured or not. The only inconvenience it puts him to is that he must report every month or six weeks for a refill, which he knows from personal experience is painless and bearable. In some cases artificial pneumothorax is more than palliative—it cures the disease radically and should therefore be applied in all cases where other methods of treatment have been tried but found wanting. Those who have treated many cases have seen many who have become self supporting at manual labor while under treatment. M. E. Rist<sup>1</sup> gives the history of a patient with an artificial pneumothorax who withstood the hardships of war unscathed.

<sup>1</sup> Presse Médicale, 1914, xxii, 692.

**Other Surgical Operations for Phthisis.—Extrapleural Pneumolysis.**

—Artificial pneumothorax is not the only method of surgical treatment of pulmonary tuberculosis. There have been suggested operations for the release of the compressed apex of the lung by the shortened first rib and ossified cartilage (p. 84); also injections of medication right into the lesion in the lung. Th. Tuffier,<sup>1</sup> in France, and Baer<sup>2</sup> and Sauerbruch,<sup>3</sup> in Germany, have developed the operation of *extrapleural pneumolysis* with a view of compressing the affected area of the lung. The object is practically the same as that of artificial pneumothorax, but with this operation only the affected part of the lung is compressed while the rest of the parenchyma is left physiologically active. It can also be applied in cases in which pneumothorax cannot, as when dense pleural adhesions prevent the injection of air or nitrogen into the pleura.

A small piece of rib is resected over the tuberculous lesion, or the phthisical cavity which is surrounded by a thick fibrous wall, and an adherent pleura which prevent its shrinkage. The lung with both sheets of the pleura is then separated from the chest wall between the costal pleura and the endothoracic fascia. The lung is then collapsed so that the walls of the cavity are brought into apposition. The space thus created under the chest wall is filled in with Beck's bismuth paste, bismuth paraffin, or plain paraffin; Tuffier uses adipose tissue, fresh or preserved. The wound is then closed properly. No general anesthesia is used, because while squeezing out the secretions of the pulmonary cavity the lungs may be flooded, and aspiration pneumonia may be the result. But local anesthesia is sufficient according to those who practise the operation.

Tuffier urges this operation even in incipient cases, saying that we should not wait in phthisis till a cavity has formed, any more than we wait in tuberculous diseases of joints till suppuration or fistulae have set in. But the modern treatment of tuberculous joint disease is rather conservative, and results are obtained which are superior to those obtained with operative treatment. It is doubtful whether the operation of extrapleural pneumolysis will ever become as popular as that of artificial pneumothorax.

**Phrenikotomie.**—Another operation which has been suggested for the cure of phthisis is resection of the phrenic nerve with a view of procuring rest of the lower part of the lung by paralysis of the diaphragm on the affected side. F. Sauerbruch<sup>4</sup> and Stuertz have done this operation in Europe and Ralph C. Matson and Marr Bisailon<sup>5</sup> have reported 2 cases in this country. It appears from the few cases reported that the operation is of no therapeutic value, if only because the diaphragm remains mobile with the respiratory movements after the operation.

<sup>1</sup> Paris Médical, 1914, iv, 231; Interstate Med. Jour., 1914, xxi, 259.

<sup>2</sup> Ztschr. f. Tuberkulose, 1914, xxiii, 209.

<sup>3</sup> Beitr. z. klin. Chirurgie, 1914, xc, 247.

<sup>4</sup> Münch. med. Wehnschr., 1913, lx, 625.

<sup>5</sup> Trans. Nat. Assn. Study and Prev. Tuber., 1915, xi, 183.



## CHAPTER XL.

### GENERAL TREATMENT OF THE VARIOUS FORMS OF PULMONARY TUBERCULOSIS.

**Incipient Phthisis.**—The treatment of the early stage of phthisis, immediately after its recognition, varies with the intensity of the clinical manifestations of the disease. We have shown that a large proportion of cases manifest a strong tendency to spontaneous cure; the disease is “aborted” within a few months. These patients need no treatment beyond stopping work, keeping regular hours, increasing the quantity of food ingested, etc. A stay in the country for a month or two is even better. In most cases of this type institutional treatment is not advisable; in fact I have seen some who were decidedly harmed by a stay in a sanatorium, where they were trained into carefully studying their disease, and impressed with the dangers of slight fever, fatigue, etc. Some have not been as industrious after the “cure” as before, though their state of health left little to be desired. With workmen having dependent families this is an important point.

It is different with patients in whom the disease manifests a tendency to acute progress; who have fever, nightsweats, cough, anorexia, emaciation, etc. These are to be given complete rest of mind and body till the acute symptoms are relieved. The best way of attaining this depends on the financial resources of the patient. The well-to-do may be treated at home or sent to private sanatoriums. The results in either case will be the same in the vast majority of cases. Under no circumstances, however, should a patient with pyrexia be sent to the country, unless he can afford to take along a well-trained nurse, and will have competent medical advice. Febrile patients, who cannot satisfy these two requirements are best treated at home, even if the home is only half-way satisfactory.

The principles of the rest cure, as well as of the treatment of pyrexia, have been given in detail elsewhere. Patients who cannot be managed at home along these lines should be sent to sanatoriums.

Patients with limited means should invariably be sent to institutions for the first few months of the disease, unless they can be moved into good homes where they may have appropriate rooms for themselves to carry out the rest and open-air treatment. But after remaining in the institutions for the period of pyrexia, they may return home where they may be cared for just as well as, and at less cost than, in the sanatoriums. Those who have no relatives or friends able and willing to give them a proper home should remain in the institutions

till the arrest of the disease is assured. As was already stated in Chapter XXXIV, the results are the same with home or institutional treatment, if the same amount of money is spent upon the patient in either case.

Reasonable patients, running only a subfebrile temperature, may be sent to the country for the first few months of the disease. Many improve to an astonishing degree, and are cured if the disease is of the milder or abortive variety. All patients should be sent out of town, preferably to the mountains, if there are no contra-indications, for the hot summer months. During the winter most phthisical patients do well in the city.

The dietetics of phthisis has already been detailed in Chapter XXXV. But it should again be emphasized that patients with a good appetite and digestion need no special diet, except that they should eat more than they had been accustomed to before the onset of the disease. In many cases an increase in the quantity of proteins and fats is desirable. Those with anorexia and indigestion are to be treated for these conditions, because good gastro-intestinal functions are the best assets of the phthisical patient. A poor appetite, if not improved by open-air treatment, should be stimulated with some of the stomachic bitters; creosote in small doses is even better for this purpose in many cases. For indigestion appropriate dietetic and medicinal treatment is to be instituted.

In the vast majority of cases medicinal treatment is not necessary in incipient phthisis, unless it is for the relief of annoying symptoms. Cough may be controlled by the administration of creosote in moderate doses. In rare cases sedatives—codein, heroin, dionin, etc.—must be given in accordance with the indications discussed in Chapter XXXVI. Anemia is to be treated with iron and arsenic. In fact most patients treated at home should be given some medication, even if it is only a placebo, and for its psychic effect alone. But there is no doubt that ichthyol, creosote and arsenic, given intelligently, exert a good influence on the course of the disease.

The treatment of complications and special symptoms, such as hemoptysis, nightsweats, emaciation, etc., has been discussed elsewhere.

Most patients in the incipient stage of the disease do well under the mode of treatment just outlined. Many will recover within a few months; in a large proportion the disease will be arrested, but they are liable to suffer from relapses sooner or later. In many the disease will continue its onward march, irrespective of the treatment applied. We then have the so-called advanced stage.

**Advanced Phthisis.**—The zeal displayed by medical men during recent years to discover and treat early cases has resulted in neglect of those in whom the lesion has advanced beyond the stage which by common consent is called incipient. Hospital wards for advanced phthisis are often attended in a haphazard fashion, and the patients

are discouraged to a pitiable extent. Patients in the advanced stages are usually told by their medical advisers to go to some distant climatic resort, irrespective of their condition. This is all wrong. There is as much hope for the average patient in the moderately advanced stage, as for a large proportion of incipient patients. Indeed we have already emphasized the fact that the prognosis in advanced phthisis depends less on the age and extent of the lesion than on the acuteness and activity of the process.

A patient with an advanced, especially cavitory lesion, owing to the fact that he has survived the incipient stage, proves that he has a certain, but variable, amount of inherent resistance against the ravages of phthisis. It is our aim to preserve, or rather to increase this power of resistance. This can only be done by proper regulation of diet, rest and exercise, and by avoiding indiscretions which are liable to produce acute exacerbations of the tuberculous process.

We therefore regulate the diet of the patient in such a manner that he will not lack in assimilable nourishment (see p. 513). The question of rest and exercise is regulated under the guidance of the thermometer and the pulse rate. In hopeful cases all efforts are to be directed at avoiding febrile exacerbations, or rendering them short lived if they occur. Many of the afebrile patients may make themselves useful in some direction. Some may even work at their occupations, provided we find that they are not harmed by activity. The fact that one has cavities in his lung does not mean that he is disabled. Patients engaged in vocations involving no undue muscular exertion may be very efficient. All should do something when strong enough to do it, but must cease all activities as soon as they feel fatigued, have fever, etc. This policy has during recent years been adopted in all the enlightened institutions for the care of the tuberculous, and the patients have benefited much more than by the previous routine rest treatment, carried out indiscriminately.

The diet in advanced phthisis is to be nutritious and of a character that will not overtax the digestive organs. At the least indication of indigestion the diet should be appropriately corrected, because, next to fever, indigestion is most liable to hurt the patient irreparably. Those manifesting a tendency to obesity, and they are not as infrequent as is commonly believed, should restrict the ingestion of fats and carbohydrates. A fat consumptive is often more miserable than a lean one.

**Medicinal Treatment.**—The average patient is not satisfied with hygienic and dietetic treatment, and when no medicinal substances are administered he is apt to be led to the belief that there is no remedy for him. But there are drugs which have a beneficial influence on the course of the disease, as was shown elsewhere (Chapter XXXVI), and medication should be administered. Considering that the patient will have to be kept under control for months, it is often difficult to allay his apprehensions, and retain his confidence till the termination of

the case. It is also a fact, to which we have already alluded, that while many remedies have an excellent influence on the disease or the patient, they retain their potency for but a short time, as a rule. The same is true of climatic resorts and institutions. The patients gain best during the first two or three months' treatment.

For these reasons medication must often be changed. Rénon's suggestion may be followed: The patient is given a course of several weeks with a certain remedy and then it is changed for another medicament administered for several weeks. The results are often remarkable: There are gains in general health, the lesion in the lung shows signs of cicatrization, and the patient is encouraged. We may thus achieve the same results as with tuberculin without incurring the hazards of this dangerous substance. A good method is to begin with ichthyol, administered as directed in Chapter XXXVI for four or six weeks; or if the patient thrives on it, it may be continued longer. For a week or two it is given in solution; for another fortnight in capsules, etc. Then we may give him creosote or one of its derivatives—creosote or guaiacol carbonate, combined with arsenic for several weeks. These substances may be given in mixtures, pills, globules, capsules, or by inhalation, as suggested by Beverly Robinson (see p. 529). The arsenic may be combined with the creosote, or given alone in the form of Fowler's solution or in pill form. Of course, if there is a tendency to hemoptysis neither the creosote nor the arsenic is to be given. The glycerophosphates are also beneficial and may be given in appropriate doses. They exert an excellent influence on the tuberculous process, promote nutrition, improve the blood picture, etc.

Medication should be discontinued as soon as there is pyrexia, though when the temperature is below 100° F. medication may and should be given.

In addition to the above there is to be given medication according to indications as revealed by the symptoms. The anorexia, night-sweats, constipation, diarrhea, etc., call for certain medicinal treatment which has already been discussed under symptomatic treatment.

In this manner the average tuberculous patient may get along very well for years. Some have very long periods of quiescence, and are only rarely laid up with acute exacerbations which need special treatment, as any acute condition. But they soon recuperate, as a rule, and again feel well for a variable period. While many survive acute exacerbations occurring at infrequent intervals, provided proper treatment is promptly instituted, in most of the chronic cases one of these acute exacerbations finally ends fatally. Many succumb to intercurrent diseases. These periods of quiescence may be obtained by judicious home treatment just as well as by institutional treatment, unless we are prepared to keep patients in sanatoriums for many years, irrespective of the activity of the disease.

Cases manifesting a tendency to progression, with acute or sub-acute symptoms and unilateral lesions, should be treated with artificial

pneumothorax. It offers immediate relief of the symptoms, and shows more striking and lasting results than any other mode of treatment of active and progressive phthisis. Many of the less acute cases are also proper subjects for pneumothorax. The indications and contra-indications are discussed in Chapter XXXIX.

Some cases show activity of the process despite the careful treatment. All efforts at raising the resisting forces are unavailing and the disease progresses to a more or less speedy termination. All we can do is to apply symptomatic treatment and to render the last weeks or days bearable and painless. The solacing effects of the derivatives of opium should not be denied these unfortunates. It is, however, one of the most common mistakes to send these patients to the country, or to sanatoriums. If such a patient has a home in which there are no infants, he may remain there. If his financial resources are limited, the proper place is a hospital for consumptives. We are at times surprised that under proper care even the most desperate case recovers, and within a few months returns greatly improved. Rarely, they even regain a capacity for working.

**Treatment of Convalescent and Arrested Cases.**—A large proportion of tuberculous patients in the advanced stages of the disease improve to an extent as to become useful at their respective occupations, although they have not been cured. They cough, expectorate, at times the sputum no longer contains any more tubercle bacilli, are more or less emaciated, but they have no fever, no tachycardia, etc. Physical exploration of the chest shows that there are cavities in the lungs, some displacements of the thoracic viscera, etc. Many of these are well able to take care of themselves and even to be efficient at some easy occupation. Under proper medical supervision they may keep on in this condition for years, even for their natural lives. It is very important that these patients have some occupation, otherwise they are liable to brood over their condition and become actual hypochondriacs. The dependent ones are liable to intrench themselves in hospitals and stay there indefinitely; when discharged they soon seek admission to another one. They are very costly to the community, as well as to those depending on them. The fact that one has a cavity in the lungs does not mean that he is disabled from working any more than one who has a chronic fistula or sinus in another part of the body. It is the intensity of the constitutional symptoms which should be the guide in these matters, and not the findings on physical exploration of the chest.

Once one has suffered from chronic phthisis of some duration, he is never cured in the anatomical sense; he is always in danger of a relapse. He should be impressed with the fact that all that was attained was an arrest of the process, and that there may be at any time a recrudescence of the disease with even greater vigor than the former attack. These arrested cases should remain under medical supervision for several years, and examined periodically; first fre-

quently, then at less frequent intervals, so that any tendency to a relapse may be checked early by proper treatment. While all efforts are to be directed toward prevention of excessive introspection and hypochondriasis, yet patients with arrested disease should be instructed as to the significance of certain symptoms, such as cough, fever, nightsweats, loss of weight, etc. During intercurrent diseases, especially catarrhal conditions of the upper respiratory passages, and influenza, they are to drop all work and take a complete rest.

A patient with arrested disease should live in a healthy part of the city, in a good home, and sleep in a room with open windows. He may engage in his former occupation, excepting the dangerous ones, but the workshop must be of the modern and sanitary type, with good ventilation, etc. When possible, workmen should become gardeners, conductors, watchmen, chauffeurs, letter carriers, etc. When feasible it is advisable that they take up farming. Well-to-do patients may move out of the city and settle for life in the country. Others may live in the suburbs, or in any country place, where they can find suitable employment. Those who remain in the city should avoid all indiscretions. The questions of marriage, pregnancy, and lactation have already been discussed.

**Acute Phthisis.**—The acute forms of phthisis are to be treated symptomatically, according to indications, as long as we have no specific for tuberculosis. In the pulmonary type of acute miliary tuberculosis careful hygienic and dietetic treatment is indicated. The nursing is of special importance, if we are to make the last days of the patient more or less comfortable. The treatment is the same as of any other acute or malignant infectious disease.

Acute pneumonic phthisis is not invariably fatal; often the patient passes the acute stage and becomes a chronic consumptive and the treatment is then the same as that given above for chronic phthisis.

During the acute stage the patient is to be kept in bed, given food suitable for a febrile case, and the indications are otherwise met as they arise. If the acuteness of the process abates, the patient remaining with an active cavity, climatic treatment may be tried. Some of these patients do very well when removed from home to some place in the country, irrespective of its location or altitude. But they usually need a nurse or an attendant. The practice of sending such patients to shift for themselves in the country cannot be too severely censured. It is unfortunate that public sanatoriums do not admit this class of cases.

**Fibroid Phthisis.**—The patient may feel well and be efficient at his occupation for many years, and the treatment at this period is purely symptomatic. It is, however, imperative to impress on them that overexertion and indiscretions are apt to activate the process.

Many patients with fibroid phthisis are well nourished during the latent or quiescent stage of the disease and need no special dietetic instructions. But we often meet with persons suffering from active

or quiescent fibroid phthisis who suffer from obesity. The dyspnea, which is a marked symptom in this disease, is more severe in the fat consumptive and it is advisable to arrange the diet so that the patient does not gain in weight excessively. Exceptionally, it is even necessary to reduce the amount of carbohydrates and fats with a view of reducing the weight of the patient. In my experience the lean, even emaciated individuals suffering from fibroid phthisis are more comfortable, and live longer than those who are obese.

In many cases the iodides are very good. The dyspnea is very often relieved, expectoration is facilitated, and the general condition of the patient improves by the administration for several months of potassium iodide or some of the newer albuminate compounds of iodine. But this remedy should not be given during febrile attacks, which are not frequent in this disease. When fever appears and is persistent, the disease differs but little from common chronic phthisis. Those who are subject to hemoptysis, and many fibroid patients suffer from recurrent hemoptysis of varying severity, should not be given any iodides. It should be discontinued immediately at the appearance of streaky sputum. In many cases with profuse expectoration, creosote gives relief.

When signs of asystole make their appearance, with dyspnea, edema, etc., appropriate doses of digitalis, strophanthus, etc., should be administered.

Fibroid patients should take frequent vacations. The mountains are not suitable for them because these patients are more short-winded the higher the altitude. It is best to send them to the plains, or the seacoast. Many do very well indeed in a desert climate, provided they can adapt themselves to the surroundings, or "rough it."

In the later stages, when fever, night sweats, cough, anorexia, etc. ensue, the case is one of advanced chronic phthisis and is to be treated accordingly.

**Pulmonary Tuberculosis in Children.**—The acute types of tuberculosis in infants are hopeless and the treatment is purely symptomatic. The infant is to be cared for as a case of pneumonia at that age. The only useful thing we can do for infants less than one year old is to prevent infection with tubercle bacilli. Once this has occurred, the prognosis is very unfavorable.

We have seen that chronic pulmonary tuberculosis of the type common in adults is practically unknown among children under ten years of age. In them the disease manifests itself as hematogenic, affecting the glands, bones and joints, and is then the province of the surgeon, though it appears from all available data that hygienic and dietetic treatment has achieved better results than the knife in these cases. The physician encounters in children disease of the tracheobronchial glands. Considering that death due to this disease is very rare, it is clear that it is bearable by most children. The only problem is whether they are all destined to develop phthisis

when reaching the age of adolescence, or later. This has not yet been solved to the satisfaction of all who are entitled to judge.

The treatment of tracheobronchial adenopathy aims at assisting nature in its efforts to preserve the child. This can best be done by doing away, as far as possible, with the unnatural method of raising children. Growing children should not be kept indoors the greater part of the day and night, but should be urged to indulge in outdoor exercises and games. Especially is outdoor life imperative when a child shows signs of tuberculous infection, or of tracheobronchial adenopathy. These children should spend the greater part of the day outdoors, and sleep in rooms with open windows. If they can be raised in the country it is much better. But in every city, excepting the parts known as the "slums," children may enjoy outdoor life and benefit by it.

It must be borne in mind that children are easily adaptable to life in cold air, and most of them can run around the street with scanty clothing during very cold days and derive great benefit. They can also be given cold spongings followed by friction with a rough towel every morning, and thus "hardened." Only in this manner can "colds" be prevented in children. Harmless in themselves, colds may, in children with tuberculous glands in the chest, activate the tuberculous process and favor an acute exacerbation of the dormant tuberculous process.

The ideal treatment of tuberculous children is to raise them all in the country. But like all ideals, it is only attainable by the favored few. The vast majority of infected children have to be raised in the cities, for obvious reasons. But society, which is largely responsible for the conditions favoring tuberculous infection, can do a great deal toward saving these children and raising them toward healthy manhood and womanhood, by providing vacations for them once or twice annually so that they may recuperate their vanishing forces and acquire resistance against the extension of the tuberculous process. In New York City this is done for a limited number of children derived from tuberculous stock by the Preventorium. In other cities in this country similar efforts have been made. But not all that need these vacations, proper food, and exercises are accommodated in any city.

If the parents of a child with tracheobronchial adenopathy can afford it they should move to the country, or to a suburb. In some cases it is feasible to send the child to be raised outside of the city lines. Many authorities maintain that it is best to raise these little patients in the mountains, or that they should be sent for frequent vacations to a high altitude. But I have seen excellent results in many cases which were sent to the seacoast, or to some forest climate. It is remarkable how quickly these children recuperate after a few weeks out in the open air away from the city.

Many of these children do not eat enough, and the emaciation resulting from the smouldering tuberculous process in the chest is



increased by the lack of nourishment. The anorexia is very often relieved by open-air life. A child in the city may not eat enough, or have an actual abhorrence for food. But as soon as it is removed to the country, the desire for food is increased; often the appetite becomes raving a few days after arrival in the country.

In those who cannot afford to go to the country the anorexia may be relieved by open-air life in the city. They should be urged to spend the greater part of the day outdoors and sleep in a room with open windows. In urgent cases there should be no schooling. The modern open-air schools are of questionable utility, especially during the winter when the bitter cold is apt to prove unbearable to both the teachers and the pupils. The child needs not only fresh air, but exercise is just as important. This keeps the child warm in the coldest day. I have very little confidence in the educational value of the open-air classes; so far as I have observed, there is hardly any study during cold days. A child run down to such an extent as to need open-air life throughout the day and night is unfit for schooling and should be taken out to the country for a few months or a season, or taken out of school for a similar period, till it recuperates, when it may resume studies.

The food of these children need not differ from that suitable for any child of the same age, but it should be plentiful, appetizing, and nourishing. It is even more difficult to place a child on a special diet than an adult. And there is no special need for such a procedure. It is, however, important to see to it that it does consume a sufficient quantity of proteids and fats. In children between two and four years of age, milk, cream and eggs supply these requirements ideally. But older children should be urged to eat meats and poultry, and butter is the best source of fat for them. It is the most assimilable form of fat that can be given to the vast majority of children. Those who do not thrive on this diet, or who will not take a sufficient amount of butter, should be given cod-liver oil. The vast majority of children take it pure, or with malt. Most of the emulsions contain very little of the oil and are nauseous.

Children with enlarged bronchial glands will almost invariably do well under this mode of treatment. It is often astonishing to watch the recuperation of an emaciated child within one or two months after being placed under this treatment. It is encouraging to watch the great improvement shown by most of the children taken from the tenements of New York City to the country or Preventorium. In some obstinate cases it is necessary to repeat the vacation twice annually for several years. Some should be kept out of town till they reach adolescence. But it should always be remembered that they all do well if properly treated; the development of chronic phthisis before the age of ten is exceedingly rare, and infrequent before the age of fifteen.

There is, however, one danger to which these children are exposed.

The endemic diseases of childhood, measles, whooping-cough, scarlet fever, etc., produce anergy, or lowered reactive powers (see p. 95) to tuberculosis. They are therefore to be guarded against these diseases. Many a child, doing well despite tracheobronchial adenitis, succumbs to bronchopneumonia complicating measles or whooping-cough. It is very difficult to carry out prophylaxis against these endemic diseases in children living in the tenements of large cities; and in those who attend school in any part of a city, where there are so many "carriers." And we cannot isolate a child from intercourse with other children for obvious reasons. This is a fact which is often not considered in this connection by those eager to do something along these lines. If after all efforts at prevention of complicating diseases have failed, and the child does develop one of them, the treatment should be very careful, and during convalescence the patient should be sent to the country for a month or so.

But infants can be shielded against infection with measles, whooping-cough, etc., because they are always in the immediate care of the mother. Infants known to have been infected with tuberculosis should be kept away from the proximity of other children who are liable to be "carriers." It is just during infancy that measles and whooping-cough are likely to do most harm when attacking a subject harboring tuberculous infection.

Medicinal treatment is not indicated in most cases, excepting where there is anemia, cough, etc. These symptoms are best relieved by the open-air treatment. But we may in many cases assist or accelerate the improvement by the administration of iron. The old *syrupus ferri iodidi* may be given in doses of 3 to 5 drops to children three years of age and more in proportion to older children. Iron tropon is another good and palatable preparation for these anemic children. The hypophosphates do good in many cases.

Children showing catarrhal symptoms, when not due to inflammatory conditions of the nose and throat, do well with creosote in small doses. It may be given in doses of from  $\frac{1}{4}$  to  $\frac{1}{2}$  drop diluted in milk. Any of the derivatives of creosote may be given in powder or in syrup form. This will often relieve a cough much more effectively than sedative drugs.

Specific treatment has been used with less success in children than in adults. It must be remembered that statistics of a number of children treated with any method, including tuberculin, are of no value if they show that of so many treated no deaths have occurred. Death due to pulmonary tuberculosis, excepting meningitis, in children over two and under fourteen years, are exceedingly rare. For these reasons, orphan asylums show such splendid results—children of tuberculous parentage do not develop phthisis while they are in the institutions. But in children tuberculin is not indicated because the psychic factor, which is the main curative factor in adults, is lacking. I can see no reason for giving tuberculin to children.

**Tuberculosis in the Aged.**—Most aged phthisical patients are emaciated and debilitated. In many nourishment cannot be given in plentiful amount because they lack teeth for mastication, and most of them suffer from disturbances in the motility and secretions of the stomach and intestines. They also have arteriosclerosis, sclerotic kidneys, and do not bear the ingestion of large quantities of proteids. Fats are apt to induce diarrhea more often than in youthful subjects.

These difficulties in the dietetics of aged consumptives may be overcome within limits by first ordering the repair of the teeth. Then they may have a diet consisting mainly of milk, cream, and cereals. Fish is also well assimilated by aged persons, and they should take it when for any reason meats are not tolerated. But as long as the condition of the kidneys is not such as to contra-indicate meats or poultry, they may be allowed in moderate quantities. Vegetables may be given as long as there is no diarrhea. While in younger phthisical patients alcohol is to be tabooed, it is different with aged patients. If they have been accustomed to alcohol it is not advisable to attempt instituting reforms at an advanced age. In some cases alcohol is even of distinct benefit, if not abused.

Old patients do not bear outdoor life as well as younger ones. The same is true of high altitude. They must have warm rooms for living and sleeping. In fact, if they can afford it they should spend the winter in some southern region. The intense cold of the winter has a very deleterious effect on them because of the defective circulation—especially the peripheral—rigid arteries, sclerotic kidneys, pulmonary emphysema, etc., with which many are affected. But they need fresh air. While they should sleep in warm rooms, the windows must be kept open.

Cardiac derangements are to be carefully treated by rest, digitalis, strophanthus, etc. Myocarditis is, however, not relieved by these remedies and, in addition to rest, small doses of nitroglycerin, frequently repeated, often have a beneficial influence. The iodides are very good in many cases and should be given in moderate doses. In many patients the dyspnea is relieved by this remedy much more effectively and lastingly than by anything else.

Fever is to be treated according to the principles discussed in Chapter XXXVIII. Most senile patients have no fever, but at times we encounter some with pyrexia of longer or shorter duration. Those in whom the fever is mild and evanescent require rest in bed until the temperature comes down to normal. Very old persons, over sixty years of age, do not bear fever very well, and must be given antipyretic treatment. Pyramidon in 5-grain doses may be administered three or four times a day.

The cough and expectoration need no treatment as long as they are not excessive. Otherwise, small doses of codein or heroin should be given. In many cases the expectoration is profuse and contains numerous tubercle bacilli. It may be greatly influenced by posture

as in bronchiectasis, and postural treatment may be attempted. But this is difficult with old persons, because of their weakness and debility they cannot withstand the vigorous cough this mode of treatment is apt to induce.

**Tuberculosis during the Menopause.**—Tuberculosis in women during the menopause is apt to be complicated by symptoms which are not seen in other phthisical patients. Considering the profound impression made by the tuberculous toxemia on the sexual sphere (see p. 238), there is no wonder that at the “critical period” tuberculous women should present special symptoms.

Many are more or less obese despite the continued activity of the tuberculous process in the lung. Dyspnea is very frequent and many complain of cardiac palpitation. Hemoptysis is very frequent, and may replace the menstrual flow, though I should hesitate before considering it vicarious menstruation. Copious hemorrhages are uncommon; I am under the impression that they are less common than among others with similar lesions. But streaky sputum and small hemorrhages are very frequent. In addition there are most of the usual symptoms of the menopause—hot flushes, headaches, etc., and profuse perspiration. Combined with the symptoms of phthisis these symptoms of the menopause make this class of patients proper subjects for special treatment.

In addition to the treatment of phthisis outlined above, the special symptoms need attention. I have had several cases in which repeated hemoptysis was stopped by the administration of the extract of the ovaries or the corpus luteum. Indeed, most of the annoying symptoms, which torture the unfortunate woman more than those caused by the tuberculous process, may be relieved by the timely and proper administration of these remedies. It is also a fact worthy of remembering that during the climacteric phthisical women do not bear the administration of tuberculin very well; most are apt to be harmed by specific treatment.

The cough and insomnia also are best relieved by the ovarian substance; sedatives and hypnotics often aggravate this condition. Though in many cases bromides and valerianates are effective.

## CHAPTER XLI.

### TREATMENT OF COMPLICATIONS.

**Pleurisy.**—Dry localized pleurisy occurring during the course of phthisis needs no special treatment, excepting to relieve the pain which is at times annoying. In mild cases external applications may suffice to give the patient comfort. Any of the belladonna plasters or a sinapism may do; while some apply tincture of iodine. The writer finds, however, that the administration of salicylates often relieves these pleural pains much better than anything else. Aspirin, in doses of from 5 to 10 grains three or four times a day, may be given in cases in which sodium salicylate is liable to derange the stomach.

In acute cases of pleurisy the pain may be very severe during the first few days before the effusion appears and may necessitate the administration of morphine,  $\frac{1}{8}$  to  $\frac{1}{4}$  grain hypodermically. In most cases it is not necessary to repeat it, but it is better to strap the chest with adhesive plaster. As soon as the effusion appears the acute pain usually ceases.

The patient is to be kept in bed as long as the fever lasts. But during the later stages he may be permitted to take mild exercises. The diet is to be given in accordance with the temperature and the tuberculous process in the lungs.

It is not advisable to make any efforts to hasten absorption of the fluid in cases of tuberculosis. The fluid may be serving a useful purpose by compressing the lung and facilitating the healing of the lesion in the same manner as an artificial pneumothorax does. On this principle effusions may be permitted to remain for months. But in case the effusion causes severe dyspnea, cyanosis, cardiac weakness, insomnia and other urgent symptoms, it should be aspirated at least partially. But even then aspiration should be left as a last resort because speedy withdrawal of the fluid and rapid expansion of the lung may awaken the tuberculous process into acute activity. The writer has observed this to happen in several cases.

It is best to first try autoserotherapy. Five to 10 c.c. of the fluid is withdrawn with an aspirating syringe and reinjected into the subcutaneous tissue. A good way is not to remove the needle after the syringe is filled with the fluid, but while withdrawing it, when its point reaches the subcutaneous tissue, to turn it parallel to the surface of the chest and to inject the fluid right then and there, as was described by the writer<sup>1</sup> elsewhere. This can be done several times on alternate days.

<sup>1</sup> Jour. Amer. Med. Assn., 1913, ix, 962.

In most cases there will be noted an increase in diuresis and the level of the fluid begins to sink, so that within a couple of weeks it may be absorbed altogether.

In cases in which autoserotherapy is of no avail and the general condition of the patient demands removal of the effusion, aspiration should be done. It is advisable not to remove all the fluid at one sitting, but to do it on alternate days, each time withdrawing a part. In many cases the pleura refills soon after tapping, and it is necessary to assist the absorption by giving a salt-free diet, and to reduce the amount of fluid ingested by the patient. Diuretin may be of assistance by increasing diuresis. But other drugs, reputed as assisting absorption of pleural effusions, as the iodides, are impotent in this regard. Emptying the bowels daily with salines, if there are no contra-indications, may assist in the absorption of the fluid.

**Empyema.**—The treatment of purulent effusion during the course of phthisis is very unsatisfactory. Some authors have stated that when the pus shows streptococci and staphylococci, the prognosis is better and resection of one or two ribs may bring about a cure, while in cases in which the pus shows the presence of tubercle bacilli, operation is futile. In the experience of the writer there was no difference from this viewpoint. On very rare occasions we meet with a case in which several aspirations of the pus cures the empyema. Similarly the writer has had 2 cases of localized and encapsulated empyemata which broke through bronchi, the pus was expectorated and the patients recovered. In the vast majority of cases we keep on withdrawing larger or smaller quantities of pus, but the chest fills up again in a short time. In some cases fistulæ form along the track of the needle, discharging pus externally.

The results of operations for empyema complicating phthisis are unsatisfactory. A simple incision for the evacuation of the pus is nearly always followed by a fistula necessitating the patient to go around with a foul smelling bandage for the rest of his life. For this reason most physicians are at present satisfied with the aspiration of the pus repeated according to indications.

Whether treated by operation or thoracocentesis, the fever usually keeps up, dropping after the removal of part of the pus, but rising again within a few days. Emaciation, night sweats, anorexia, diarrhea, etc., keep on; amyloid degeneration of the viscera, notably the liver spleen, kidneys, and intestines, develops and the patient sooner or later succumbs to exhaustion.

The suggestion of some authors that after removing the pus nitrogen should be inflated into the pleura has been tried by the writer, not found to offer any advantages, and was abandoned.

**Spontaneous Pneumothorax.**—In the treatment of this complication we must consider whether this accident may not ultimately turn out of use by collapsing the lung and thus facilitating the healing process as the artificial variety often does. This is exceedingly rare;

still now and then we meet with a case in which a spontaneous pneumothorax is followed by improvement in the symptoms of the original disease.

The acute onset with shock, pain, dyspnea, etc., demands active treatment. The indications are clear: The patient is to be relieved of the urgent and menacing symptoms, his heart is to be stimulated, etc., which is best done by a hypodermic injection of morphin. But if the patient is not calmed, and the dyspnea is urgent, thoracocentesis is to be performed. This is often the only means at our command to relieve the extreme and agonizing dyspnea. Tapping the air in the affected pleural cavity gives prompt relief, though unfortunately only of short duration in most cases. Plunging a hypodermic needle into the affected side is sufficient, because the expiratory pressure within the pleura is greater than that of the external atmosphere. It is good to attach a rubber tube to the needle by one end, while the other is placed in a pail of water, thus forming a water valve which permits the free exit of the air from the chest, but prevents its return.

If the relief thus obtained is only transitory, the operation is repeated; in some cases it may be necessary to repeat the tapping four, five, or even seven times during the first day. Some have tried to obviate this by inserting a cannula and leaving it in the chest wall for several hours or days; the rubber tube all the time in the water. But I have found it very difficult to retain the needle in place and to keep it aseptic. For this reason I prefer to make several punctures as the urgency of the symptoms demands.

Many theoretical objections have been raised against tapping the chest in these cases. But one has only to witness a case in which the agonizing pain and air hunger are promptly relieved by tapping, to appreciate that this is the only measure which gives relief. As in urgent cases of any kind, theoretical considerations are left till the menacing symptoms are under control. In fact, after one tapping the patient begs for another when the dyspnea returns.

We meet with cases in which the embarrassment of the circulation and respiration continues in spite of repeated tapplings, and the prognosis is gloomy. The causes are not primarily mechanical, but physiological. The opposite lung is congested and the circulation is thereby more embarrassed than by the displacement of the mediastinum. In these cases we may try oxygen inhalation and cupping all over the posterior aspect of the chest. Some use wet cups or venesection to relieve the right ventricle which is becoming paralyzed from extreme overdistention. "I have no doubt," says West, "that life might be sometimes saved by timely venesection and it is certain that bleeding is not as much employed in these urgent cases as it ought to be."

The heart section is to be sustained by large doses of strychnia, digitalis, spartein, or camphor.

In milder cases, especially those in which the pneumothorax is only partial and the symptoms are not so urgent, the treatment is less

vigorous. The dyspnea, pain, and distress are usually controlled by a dose of morphine hypodermically, and within a day or two the patient feels quite comfortable.

The after-treatment, if the patient survives three or four days, is that of the underlying tuberculous process in the lungs. Inasmuch as the pneumothorax with its sudden onset and agonizing symptoms often leaves the patient in a debilitated condition, rest and proper feeding are to be enforced. In rare cases the pneumothorax; acute and menacing as it was at the onset, turns out to be "providential," as some French authors say. The collapsed lung is given an opportunity to heal and recovery may take place ultimately. Some recommend that in such cases the pneumothorax should be continued by injections of nitrogen in the approved manner.

After the menacing symptoms have abated the patient, regaining his strength and composure, provided he has no fever, may be permitted to leave his bed and take mild walking exercises. We know now from experience with artificial pneumothorax that one can do considerable exercise or even work when one pleural cavity is filled with air and the lung collapsed. But a spontaneous pneumothorax is not always closed and exercises may cause some of the morbid secretions to enter the pleura through the fistula and cause pyothorax.

**Hydropneumothorax.**—The treatment of effusion into a pleural cavity filled with air is conservative, just as that of pneumothorax. The fluid is absorbed sooner or later spontaneously. We now have experience with this condition in cases with artificial pneumothorax. As long as there is no fever or dyspnea the patient may be allowed considerable exercise. But in case the intrathoracic pressure becomes high and produces dyspnea when the patient is at rest, the pressure must be reduced. This can be done by withdrawing some of the air or fluid. The latter is the best. With an aspirating apparatus a part of the exudate is withdrawn. In many cases the operation has to be repeated. In favorable cases this withdrawal stimulates the absorption of the rest of the fluid. In several cases I have had good results with autoserotherapy (p. 613).

**Pyopneumothorax.**—The treatment of this complication is very unsatisfactory. Operative interference has not given encouraging results. At best, a fistula is left in the chest which discharges pus indefinitely. The ultimate result is worse than when only tapping of the pus is resorted to. The indications, therefore, are to aspirate the pus at frequent intervals with a view of keeping the patient afebrile as far as possible. The bacteriological findings have no influence on the prognosis and treatment, as has already been stated when speaking of empyema complicating phthisis.

**Laryngeal Tuberculosis.**—Many cases of tuberculous laryngitis show a strong tendency to spontaneous cure, especially in patients whose lung lesion also manifests a tendency to improvement. In fact the progress of the lesion in the larynx goes hand in hand with the



progress of the lung lesion, though the physical signs of the latter are apt to be obscured by the former. This is clearly seen in cases in which the induction of a therapeutic pneumothorax is effective in curing the patient. If there has been a laryngeal lesion it often shares in the general improvement of the patient.

In my experience local treatment is not often effective in enhancing cicatrization of laryngeal lesions. When carried out vigorously, it is apt to do harm. The application of local escharotics and cauterization has been harmful in the long run, or of no benefit in the vast majority of my cases. As has been pointed out by St. Clair Thomson,<sup>1</sup> lactic acid, which is the favorite drug used by laryngologists, is unavailing except in strengths of 50 per cent. or more. Hence sprays of 2 per cent. are nothing but irritating. Frequent applications are also irrational, the object being to produce an eschar which does not separate for one to three weeks. When the slough is detached a healing ulcer is exposed; but there are generally deeper deposits requiring a repetition of the cauterizing process, so that four to twelve applications may have to be spread over as many months. The use of a 20 to 25 per cent. solution of argyrol, or a 2 per cent. solution of methelene blue for local application, as advised by Fetterolf, is less likely to be painful or harmful. Where the mucous membrane is unbroken no local application of drugs does any good.

In a few cases I have seen excellent results when the patient ceased talking altogether, thus affording perfect rest to the larynx. But it must be done thoroughly. The patient should have a pad and pencil and carry on all conversation in writing. In two cases, both women, in whom this treatment was carried out perfectly, the laryngeal lesion healed. There are, however, few patients who want to submit to this treatment for a long time. In patients with advanced and active lesions in the lungs, there is no reason for trying it, because they are doomed anyway.

As has been shown by Fetterolf,<sup>2</sup> there is one form of the disease in which unlimited use of the voice is advisable, this being the variety in which the vocal cords are the only parts of the larynx involved. This is commonly called the "chorditic" form, the cords appearing slightly congested and having on their upper, and to a slight extent on their mesial aspect a number of reddish granular growths. These are possibly sometimes submucous tubercles, but more frequently are distended mucous glands with their duct orifices occluded. Vocal exercise aids in clearing up the condition, and it is in this form that improvement of the voice so frequently follows an acute coryza.

In all cases with dysphagia palliative treatment must be applied. We may try to obtain relief by laryngeal insufflations of 3 to 5 grains of orthoform or anesthesin. It is only effective when there is ulceration and the powder remains on the ulcer. If given about one hour before

<sup>1</sup> Diseases of the Nose and Throat, New York, 1912, p. 606.

<sup>2</sup> Hare's Modern Treatment, Philadelphia, 1911, ii, 402.

the main meal the patient may be comfortable for a whole day. The following formulæ may also be used:

R—Orthoformi . . . . .	gr. xxx	2.0
Iodoformi . . . . .	gr. xxx	2.0
Mentholi . . . . .	gr. vj	0.4
M. S.—Insufflate a few grains one hour before meals.		
R—Cocaine hydrochloridi . . . . .	gr. x	0.7
Morphinæ hydrochloridi . . . . .	gr. ij	0.1
Mentholis . . . . .	gr. xv	1.0
Iodoformi . . . . .	ʒij	8.0
Acidi borici . . . . .	ʒij	8.0
M. S.—Insufflate a few grains one hour before meals.		

The application of these powders is to be made with special insufflators. They are designed so that the spray goes vertically downward, not backward into the pharynx.

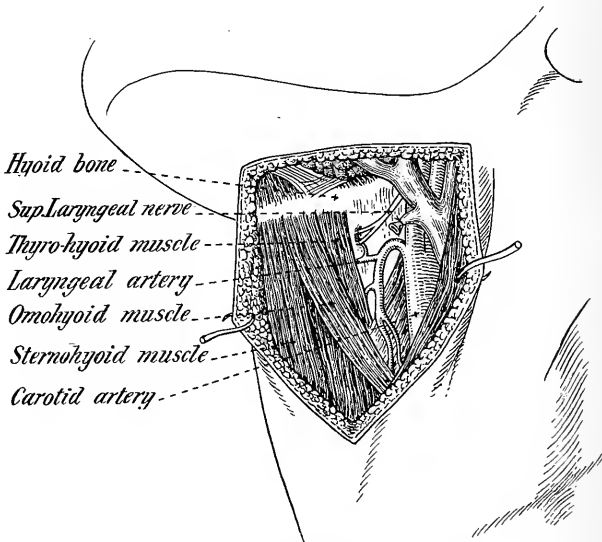


FIG. 90.—The thyrohyoid region. (Grivot.)

In some cases the dysphagia is severe and not at all influenced by the application of remedies locally. Injections of alcohol into the superior laryngeal nerve may then be tried. Relief from pain may be obtained lasting several weeks. Rudolf Hoffmann was the first to suggest this mode of treatment. The technic of the injection is thus given by J. Dundas Grant.<sup>1</sup>

Place the patient in a horizontal position and, with the thumb of the left hand, press the sound side of the larynx toward the middle line so that the affected half projects distinctly; the other fingers of the hand lie on this. The index finger enters the space between the

<sup>1</sup> Lancet, 1910, i, 1754.

thyroid cartilage and the hyoid bone from without until the patient announces that a painful spots had been reached. With a little practice one arrives at it at the first go-off, when one has become familiar with the topographical relations. Now the nail of the index finger is placed on the skin (which has been previously disinfected) in such a way that the point of entrance for the needle lies opposite its middle. The needle is pushed in for about 1.5 cm. and this distance is marked off on the needle perpendicular to the surface of the body. According to the thickness of the subcutaneous layer of fat, the perforation has to be more or less deep. The needle is then carefully moved so as to seek a spot at which the patient states that he feels pain in the ear. The syringe filled with 85 per cent. alcohol warmed to

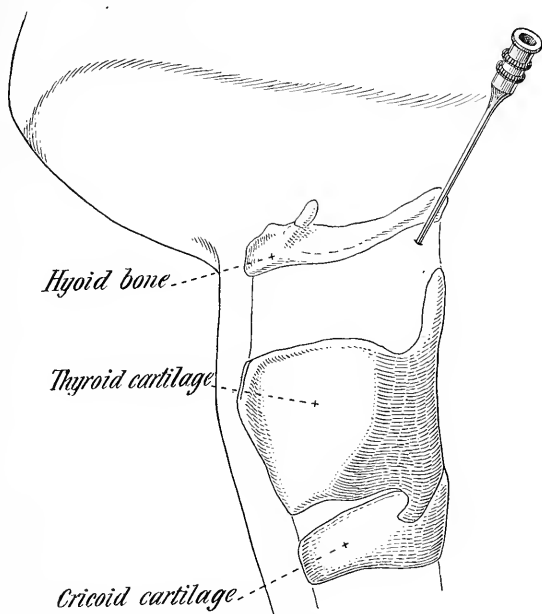


FIG. 91.—Space where to insert the needle for producing anesthesia of the superior laryngeal nerve. (Celles.)

the temperature of 45° C. (113° F.) is screwed on to the handle and the piston is then slowly pressed down. The patient now feels pain in the ear, the passing off of which he indicates by raising his hand. During the operation he has to avoid both swallowing and speaking; if, however, he makes a movement of swallowing we must follow the movement of the syringe with a light touch. The injection is kept up until no further pain occurs in the ear; then the needle is removed and collodion or sticking plaster is placed on the spot of the injection without pressure. The needle employed should be one with a point bevelled off much more obtusely than in an ordinary hypodermic needle, so as to avoid the risk of puncturing a vessel.

I have tried this method in many cases and obtained relief for the patient in about 50 per cent. Failures are due to missing the nerve, which is unavoidable in many cases.

There are cases in which all the above fail to relieve the sufferer and all we can do is to give large doses of anodyne drugs. In some we may obtain relief by helping the patient in the following manner while he eats: A trained person stands behind the patient and makes firm and even pressure at the angle of each jaw at the moment of swallowing. Another way is known as Wolfenden's position: The patient lies prone over the bed with the face over the end and sucks the nourishment through a glass tube from a cup on the floor. These manoeuvres seem cumbersome, to say the least, but when having a patient under our care who cannot swallow even water without severe pains in the throat, we are ready to try anything.

There remains yet to mention the various operations of curettage and cautery which laryngologists perform in these cases. Some employ direct laryngoscopy while operating, but this is not only violent, but the results have been disastrous in all the cases that have been done for me. In advising operation to a patient of this class we must first ascertain the general and the local condition of the lungs. In case the prognosis is poor from the general condition, there is no reason for operating. I always object to operations in febrile and cachectic patients.

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Pulmonary tuberculosis



